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## SYMPOSIUM ON RELIEF OF PAIN

IN a recent number of the Medical Clinics there was a Symposium on Pain. In this issue we have carried the thought a little further and have gathered a group of clinics on the Relief of Pain. It has been our endeavor to cover the regions of the body in which the relief of pain is the problem of the general practitioner.

The following clinics are included in this Symposium.

Abraham Ettleson RELIEF OF HEADACHES FACIAL NEURALGIA, GLOSSOPHARYNGEAL NEURALGIA SUPERIOR LARYNGEAL NEURALGIA OCCIPITAL NEURALGIA PAIN FROM SLUDER'S NEURALGIA.

Robert O. Ritter RELIEF OF LUMBAGO AND SCIATICA

Louis T. Cury TREATMENT OF EARACHE.

Charles W. Freeman PAIN IN THE DENTAL FIELD

Walter R. Fischer RELIEF FOR PAINFUL FEET

G. K. Fenn PAIN SIMULATING THAT PRODUCED BY CORONARY DISEASE.

N. C. Gilbert TREATMENT OF ANGINA PECTORIS.

Lowell D. Snorf PAIN IN THE ABDOMEN CLINICAL SIGNIFICANCE AND CONSIDERATION OF RELIEF

J. P. Greenhill RELIEF OF PAIN ARISING IN THE FEMALE PELVIS.





## CLINIC OF DR ABRAHAM ETTLESON

LOYOLA UNIVERSITY

### RELIEF OF HEADACHES, FACIAL NEURALGIA, GLOSSOPHARYNGEAL NEURALGIA, SUPERIOR LARYNGEAL NEURALGIA, OCCIPITAL NEURALGIA, PAIN FROM SLUDER'S NEURALGIA

#### RELIEF OF HEADACHES

THE clinic today concerns itself mainly with the relief of headache due to migraine. It is to be understood that the following outline of treatment is intended only for those patients in whom a diagnosis of migraine is made after all other causes of headache have been excluded. For a general discussion of migraine the reader is referred to the literature on that subject.

In order to obtain relief from this distressing affliction, it is necessary to avoid possible precipitating or exciting causes of attacks of migraine such as fatigue, mental or physical, continued worries, fears and excitement, indiscretions of eating, drinking or smoking, and exposure. In a positive direction, the sufferers from migraine should adhere to a regular hygiene of living with regulation of diet and bowels, abundance of fresh air, sleep, and light daily exercise. On general medical principles physical defects should be corrected, although their correction may not bring relief from the headaches. Likewise the removal of foci of infection and the cure of constipation may fail to produce the desired results.

With recent advances in allergic and endocrine disorders, treatment for the relief of migraine has been undertaken by specialists in these fields with reported variable success.

Among the allergic disorders food disagreements have been blamed for the periodic headaches, for which skin tests are made, and "elimination diets" are given, or protein desensitization with peptone or other nonspecific protein is done. In the endocrine field various drugs from the selection of organotherapy are employed particularly theelin one ampule every other day for one week before regular menstrual periods, and placental hormone (emmenin), an emmenagogue, or gynergen before menstruation. The rationale for the use of these drugs is the frequent association of attacks of migraine with the menstrual period. It is wise not to promise the sufferer relief from these measures, for failures from these forms of treatment are common, and promise unfulfilled, confidence is lost.

Regarding the use of drugs for the relief of migraine many have been tried, but none is specific for the disease. Among them the two which have received the most credit for affording relief are fluid extract of *Cannabis indica* in the largest dosage tolerated, starting with 2 minims every four hours, and sodium thiosulphate 15 grains intravenously. The former probably produces its desirable effects by depressing the sensory pathways in the spinal cord, and, in large doses, producing sleep. The favorable influence of thiosulphate on headache is not understood. The bromides, luminal, calcium salts, salicylates, and caffeine have been tried, each alone or in combination, with varying results, but on the whole they have not been successful. Morphine is to be avoided, or used only as a last resort, because of the danger of addiction.

Ligation of the middle meningeal artery on one or both sides is being done today after all palliative measures have been tried and failed. In the few reported operated cases and in those known to me, the ligation effected relief from the severe headaches. The operation which is only slightly more than a subtemporal decompression, can be done under local anesthesia in the upright position. It is a relatively simple and short procedure, practically entirely devoid of risk.

The reason for the success of this procedure is explained by the theory that migraine headaches are caused by vaso-

motor spasm of cerebral and meningeal vessels brought on by an irritative affection of the cervical sympathetic ganglia. This supposition is rendered plausible by the recent demonstration of nerve fibers in cerebral vessels. Spasms of the latter produce the various visual disturbances, aphasia, paresthesias, and psychic phenomena of migraine, while constriction of the meningeal vessels is thought to be the cause of the severe hemicrania through the involvement of the sympathetic plexus encircling the latter. Hence, severing the artery destroys the sympathetic plexus around it, thus preventing spasm of the vessel. On the other hand, avulsion of the sensory root of the gasserian ganglion on the affected side has not brought relief from migraine headaches.

For the relief of an attack "nearly every patient has his own pet remedy" (P. Bassoe). A hot mustard foot bath, or a warm full bath, followed by rest in bed in a quiet, darkened room with warm compresses to the head and eyes may afford relief from the intense headache. One or more of the coal tar products—phenacetin, aspirin, pyramidon, each in 5 grain doses, and inhalation of the contents of a pearl of amyl nitrite may be effective.

#### RELIEF OF FACIAL NEURALGIA

We shall consider here chiefly the relief of trigeminal, or, as commonly called, trifacial neuralgia. All other causes of pain in the face must be looked for and excluded before diagnosis of *tic douloureux* is made. In this regard it is particularly important to distinguish between psychalgia and true paroxysms of trigeminal neuralgia. In the latter condition the pain always occurs in paroxysms and is limited to the true anatomical distribution of the fifth nerve on one side, never spreading across the midline or beyond the nerve area, while in the former the pain may be continuous and located anywhere in the face, head and neck, or shoulders.

Having determined that the pain in the face is due to major *tic*, relief may be obtained by one of several procedures. Since one of the cardinal features of trifacial neuralgia is the presence of hyperesthetic areas or trigger zones on the face,

scalp, or in the mouth which when touched set off a paroxysm, the sufferer will have learned from painful experience to leave these tender points absolutely untouched. He will therefore refrain from rubbing his eyes, touching, washing and wiping his face or lips, combing his hair, brushing his teeth, laughing, talking, blowing his nose, sneezing, coughing, or moving his jaw, according to the branch of the fifth nerve affected. He will also have learned to avoid cold drafts and chill, excitement and noises.

As in migraine, here also on general medical principles all possible local causes of pain in the face, as infected teeth or nasal sinuses, should be treated, although no relief may follow. By the time the patient will have drifted into the hands of a neurologist or neurosurgeon, the victim already will have sacrificed most or all of his sound teeth and submitted to one or more operations on his nose and sinuses without avail.

For temporary relief of pain deep inhalation of 20 to 30 drops of trichlorethylene (chlorylen) poured on a piece of gauze while patient reclines on one side is recommended. This drug acts selectively on the fifth nerve. All other drugs including morphine are worthless in the treatment of this affliction.

Roentgen radiation over the gasserian ganglion has lately been tried, but the fact that it has not displaced other measures after several years of trial indicates the limited efficacy of this procedure. However, postherpetic neuralgic pain is relieved by this method but not by operation.

Alcohol injection into the nerve trunks was the best known method of treatment until surgery began to be employed for the relief of facial pain. This method is still useful in many cases, especially in those in whom surgery is contraindicated. It is for the benefit of these cases that the technic of alcohol injection is here given in detail. Alcohol injection offers only temporary relief, the pain recurring in from six months to a few years. It is always advisable to inform the patient about the numbness of the face that follows injection.

The *modus operandi* of alcohol when injected into a nerve

explains the reason for only temporary relief from pain. Alcohol when injected into the nerve causes destruction by coagulation of the nerve fibers at the site of injection followed by degeneration of the nerve elements *below* this point to the periphery. The injection, which is excessively painful over the area supplied by the nerve, produces anesthesia almost immediately. So long as the nerve fiber *above* the point of injection and its nerve cell of origin within the ganglion are intact, a new nerve fiber will commence to grow downward from the cell body during the process of nerve regeneration, and by this means the damaged nerve is renewed and its conduction powers are re-established after an interval varying from six months to two years. Injection of the gasserian ganglion may offer permanent relief, but it is fraught with great danger except in the hands of a highly skilled operator. The alcohol might enter the optic nerve and produce blindness, or the oculomotor nerves and cause paralysis, or the cavernous sinus resulting in thrombosis. It makes later resection of the sensory root much more difficult. It may lead to corneal ulceration just as resection of the sensory root, and hence has no advantage over the latter procedure. In short, injection of the gasserian ganglion is not recommended.

Furthermore, an excellent knowledge of the anatomy of the trigeminal nerve is necessary to be successful with alcohol injection for the relief of tic douloureux. The depth to which the needle must be inserted in the tissues is very important, and can be determined by placing a small piece of rubber on the needle (Fig. 115) before insertion to the estimated depth of the nerve to be injected. The surest sign that the needle is in the correct position is the occurrence of pain in the area supplied by that nerve. The point of the needle must puncture the nerve trunk so that the alcohol actually penetrates among the nerve fibers.

The needles used are of stainless steel and flexible, like the kind employed in local infiltration anesthesia. The skin at the site of injection is made antiseptic in the usual manner. The alcohol is *never* injected into the nerve until anesthesia

has resulted from injecting a few drops of 2 per cent novocain. The syringe with novocain is then replaced by one with 95 per cent alcohol and the nerve is injected with it one drop at a time until the area of the nerve is totally anesthetic to pain, and there is no longer felt a burning sensation in the cutaneous distribution of the nerve.

**Neuralgia of the First Division**—*Technic for Injection of the Supra-orbital Nerve*—(Figs 111, 112) The supra-

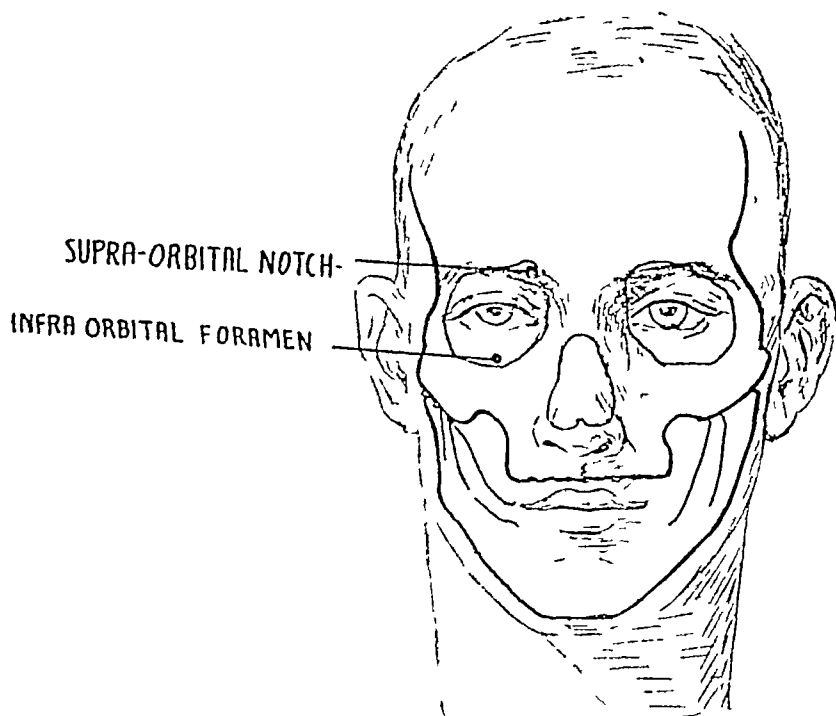


Fig 111—Points of injecting the supra-orbital and infra-orbital nerves

orbital notch is marked by the index fingernail of the left hand pressed upon it while a small flexible needle is slipped along the fingernail down into the notch, just below the eye-brow, at a distance of about 1 inch from the middle. As the point of the needle hits the bone, the patient will experience a sharp pain up to the top of the head. This is the sign that the needle is in the correct position. Now 2 or 3 drops of 2 per cent novocain solution are injected, and the sensation

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of the forehead is tested by a pinpoint. If anesthesia in this area has resulted, the syringe with novocain is replaced by one with 95 per cent alcohol and about 10 drops are injected slowly, 1 drop at a time. There should result anesthesia of the forehead and top of the head up to the midline, except

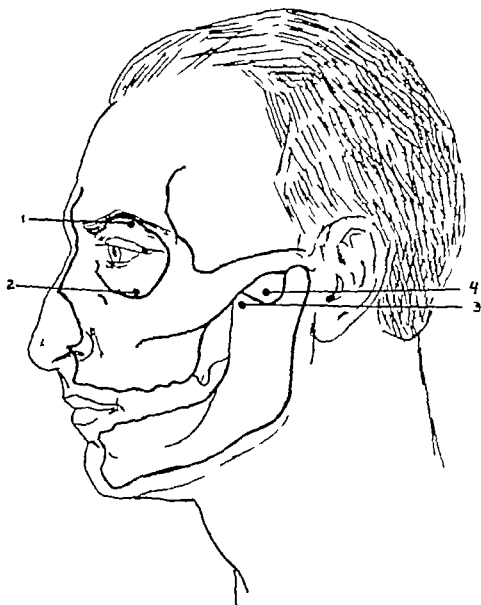


Fig 112.—Points of injection of supra-orbital infra-orbital maxillary and mandibular nerves. 1 Supra-orbital notch 2 infra-orbital foramen 3 coronoid process of mandible 4, notch of mandible

the central triangle of the skin between the eyebrow and midline which is spared, because it is supplied by the supra-trochlear nerve.

**Neuralgia of the Second Division.**—(a) *Technic of Injection of the Infra-orbital Nerve*—(Figs 113, 111, 112)

The infra-orbital foramen lies about 1 inch from the midline. A long needle is inserted at a point about one-fourth of an inch outside and above the angle of the ala nasi, the insertion being made down to the bone in an upward and slightly outward direction. It is important to keep the left forefinger firmly pressed against the rim of the lower margin of the orbit, to insure that the needle does not slip over the orbital margin and so penetrate the orbit, where serious damage might be done. The patient will complain of pain in the nose and

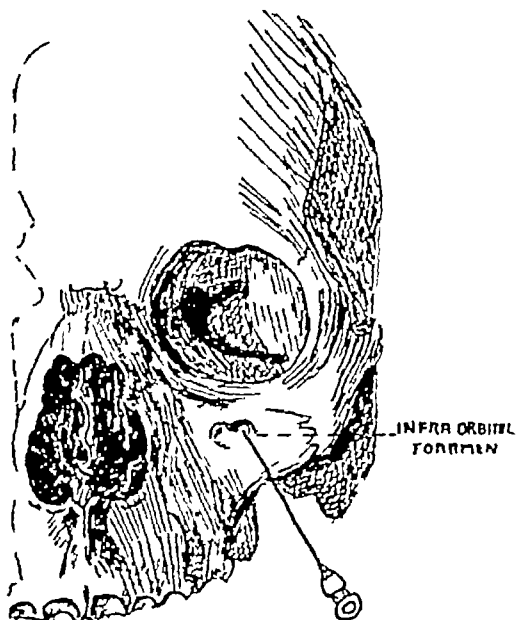


Fig. 113—Anatomical view showing injection of infra-orbital nerve

upper lip as soon as the needle strikes the nerve. Three or 4 drops of 2 per cent novocain solution are then injected, which will be followed by anesthesia of the upper lip, lower eyelid, cheek, and side of nose. Then 1 cc of 95 per cent alcohol is slowly injected, a dressing being held firmly pressed against the cheek close to the needle to prevent swelling.

(b) *Technic of Injection of Second Division at Foramen Rotundum*—(Figs 114, 115, 112) Injection of the second and third divisions at the foramen rotundum and foramen

ovale, respectively, and gasserian ganglion should be attempted only by the skilled physician. A skull with mandible in place always should be before the operator for guidance by landmarks during injection of the second or third division. For injection of the second division it is well beforehand to mark off on the needle  $4\frac{1}{2}$  to 5 cm from the point upward. The method of approach to the foramen rotundum and foramen ovale is through the outside of the cheek.

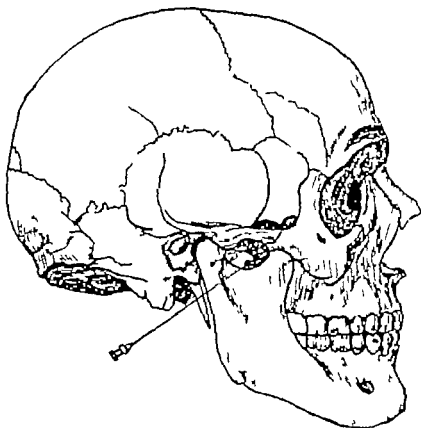


Fig 114—Lateral view of skull showing anatomical landmarks for injecting the second and third divisions

First feel the coronoid process of the mandible, then insert a long, flexible needle behind this process. Now direct the needle slightly forward at an angle of from 15 to 30 degrees, and upward at an angle of about 15 degrees.

The object is to hit the external pterygoid plate (Fig 116), and then to manipulate the needle forward until its point is felt to slip in front of the anterior edge of the plate, where the nerve will be found at a slightly greater depth. If the nerve is not found on the first attempt the needle must be

partially withdrawn and its angle of direction altered slightly upward or downward, until the nerve is found. At a depth of 5 cm the needle should be in the foramen rotundum. The penetration of the nerve by the needle will be indicated by pain in the distribution of the second division in the upper jaw, teeth and gums. The injection first by novocain and then by alcohol is made as for the third division injection (See below.)

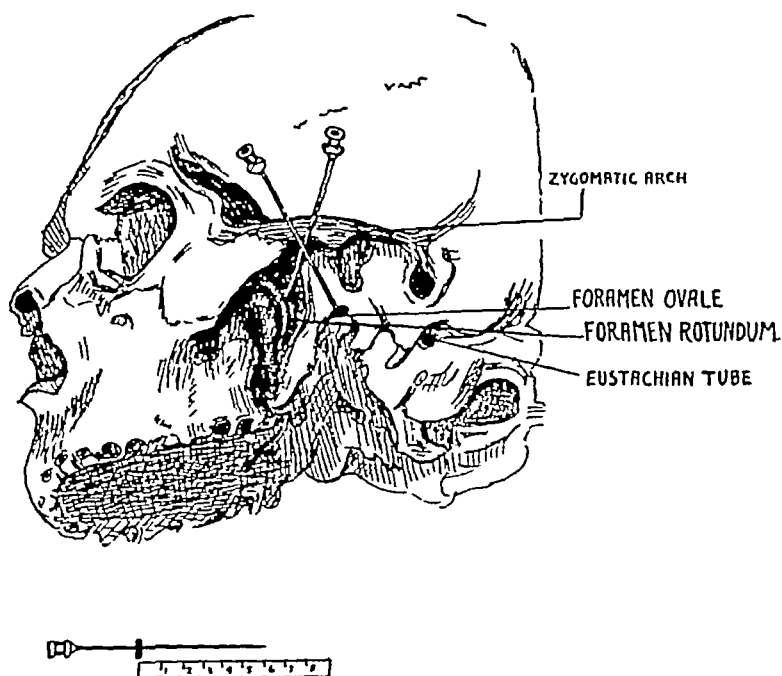


Fig 115—Anatomical view showing the foramen rotundum and foramen ovale at sites for injection of the second and third divisions respectively. Piece of rubber marks the length of needle to be inserted down to the nerve.

**Neuralgia of the Third Division**—(a) Peripheral injection of this division is made in the mental foramen, through the cheek or into the inferior dental nerve on the inner side of the ramus of the lower jaw (Figs 116, 117.)

(b) *Injection of Third Division at Foramen Ovale*—(Figs 114, 116, 115, 112.) A long flexible needle is inserted in the middle of the zygomatic arch, through the sigmoid notch of the mandible, about 1 inch forward from the middle of the

external auditory meatus Push the needle inward pointing it slightly upward and backward Pain referred to the ear indicates that the eustachian tube has been touched because the needle is too far backward Withdraw it a little and push it further forward Pain felt in the throat and back of the tongue means that the needle has been pushed too far By the method of trial and error the third division can even-

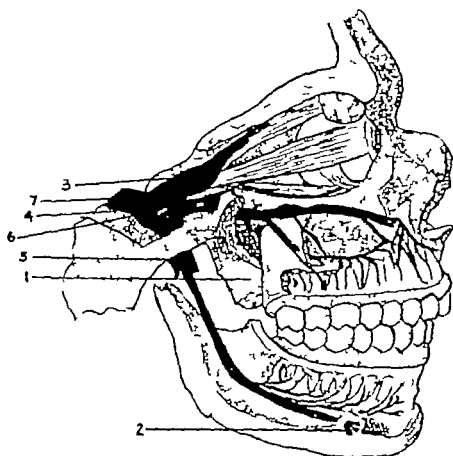


Fig 116—Anatomy of trigeminal nerve. 1 External pterygoid process of sphenoid bone 2, mental foramen 3 ophthalmic division 4 maxillary division 5 mandibular division 6 gasserian ganglion, 7 sensory root of gasserian ganglion

tually be found As soon as the needle strikes the nerve, the patient complains of pain in the lower lip, chin, or tongue Inject 2 or 3 drops of 2 per cent novocain and test these parts for anesthesia When this appears change the syringe to 95 per cent alcohol and inject slowly a drop at a time As the alcohol is injected, a burning sensation is felt in the lower lip, cheek and chin, and a wooden feeling spreads along the

lower jaw and in front of the ear. The burning sensation soon passes off whereupon more alcohol is injected, 2 or 3 drops at a time, until the anesthesia is complete and no more burning is experienced. The pain then ceases at once.

If the burning pain spreads to the nose, cheek, and eye, and anesthesia appears over the upper two divisions of the nerve, this indicates that the gasserian ganglion has also been injected.

Successful injection results in anesthesia to pain and touch on the side of the lower lip, inside of cheek, lower jaw, teeth, gums, and tongue, on a strip of skin extending from the lower

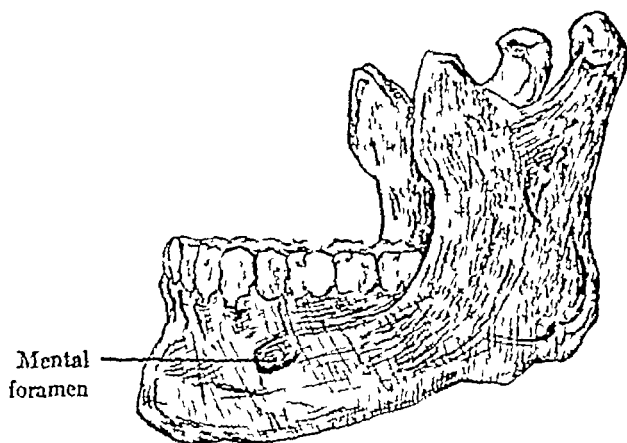
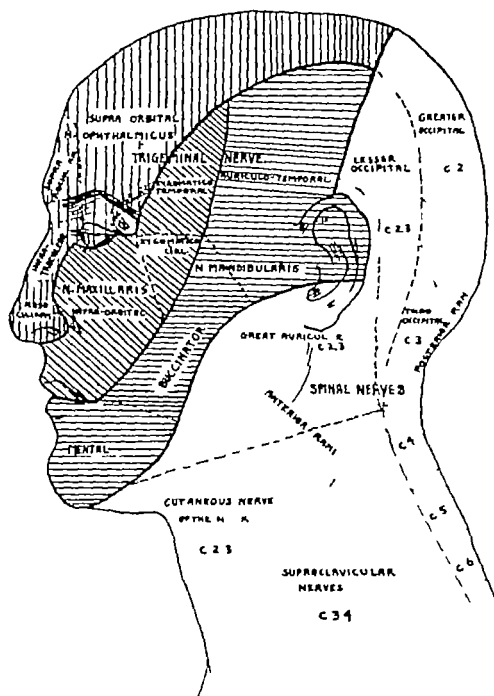


Fig 117—Mandible showing mental foramen

angle of the mouth toward the temple, and in the external auditory meatus and tympanum (Fig 118). There will also be paralysis of the masseter, temporal and pterygoid muscles, so that the lower jaw when opened widely will be pushed toward the side injected.

Following an injection the patient may complain of deafness and intense vertigo and he may vomit and have facial paralysis and coarse nystagmus due to leakage of the alcohol toward the cerebellopontine angle. This can be prevented by making the injection very slowly.

The chief danger, arising from the unintentional injection of the gasserian ganglion, is corneal ulceration. This can be



- OPHTHALMIC DIVISION  
 MAXILLARY DIVISION  
 MANDIBULAR DIVISION

Fig 118—Sensory area of head showing the three divisions of the fifth nerve

recognized early by the steamy ground glass appearance of the cornea, with circumcorneal injection. If this is observed, a drop or two of atropine in the eye with closure of the lids



by a firm bandage for twenty-four hours may prevent further ulceration. Whenever the corneal reflex is abolished after an injection of the foramen ovale, the eye requires special attention, by shielding it with an eye shield or crystal and irrigating with 2 per cent boric acid lotions and instillation of 10 per cent argyrol.

Avulsion of the sensory root of the gasserian ganglion, also referred to as subtotal resection of the root, or retrogasserian neurectomy, offers the only permanent relief from trigeminal neuralgia. In this operation the sensory root fibers (Fig 116) of the second and third divisions are destroyed never to regenerate functionally. The ophthalmic division and the motor root of the fifth nerve are left intact whenever possible to avoid the risk of keratitis and paralysis of the jaw. Nearly the entire operation can be done under local anesthesia in the sitting position, via the subtemporal route. Sufferers from trifacial neuralgia who also have cardiac decompensation and auricular flutter require preliminary digitalization, otherwise the operation may be a success but the patient will succumb to cardiac failure.

The surgical risk from this operation is small indeed, but the greatest danger is ulcer of the cornea which may so resist all treatment that enucleation may have to be done. The patient should be informed of this danger beforehand. Usually, however, this hazard will not deter him from submitting to surgery, in his desperate search for relief from pain.

#### RELIEF OF GLOSSOPHARYNGEAL NEURALGIA

The relief of pain from glossopharyngeal neuralgia is required infrequently, as this type of neuralgia is uncommon. It is not to be confused with trigeminal or superior laryngeal neuralgia.

After all causes of pain in the throat, such as organic diseases of the pharynx, neck, posterior cranial fossa, etc., have been eliminated one is justified in considering the pain due to glossopharyngeal neuralgia and in instituting treatment for this condition.

There are three methods of treatment for the relief of this type of neuralgia. Alcohol injection of the nerve near the jugular foramen, neurectomy of the ninth nerve as high up in the neck as possible, and intracerebral section of this nerve.

Alcohol injection in the region of the jugular foramen is not to be attempted by the inexperienced, as the danger of paralysis of the bulb is great. For the injection the needle is inserted in front of the tip of the mastoid, from where it is passed inward and slightly upward to a depth of  $1\frac{1}{2}$  inches. When the needle strikes the nerve, pain will be felt in the lateral region of the throat radiating to the eustachian tube and behind the ear.

Injection is then made with 2 per cent novocain followed by 95 per cent alcohol, described under trigeminal neuralgia, with resulting relief of pain in, and anesthesia of, the pharynx, and loss of taste on the posterior third of the tongue.

This method and neurectomy of the ninth nerve may afford relief varying from six months to two years, after which the pain recurs. Intracerebral section of the glossopharyngeal nerve effects a permanent cure from the pain.

#### RELIEF OF SUPERIOR LARYNGEAL NEURALGIA

Superior laryngeal neuralgia is even more uncommon than the glossopharyngeal type, from which it must be differentiated. The former must not be mistaken for tuberculous or malignant disease of the larynx and epiglottis, or for the gastric crises of tabes.

In alcohol injection for the relief of pain from this form of neuralgia, only the internal branch of the superior laryngeal nerve is injected. With the left index finger on the greater cornu of the hyoid bone (Fig. 119), the needle is inserted in the midline between the superior laryngeal margin and the hyoid bone and is carried laterally through the thyrohyoid ligament, which offers considerable resistance toward the cornu of the hyoid bone. The internal branch is then located by probing with the needle until pain is felt in the side of the neck shooting up to the ear, and from the side of the

thyroid cartilage darting up to the angle of the jaw. Successful injection with novocain and alcohol produces anesthesia

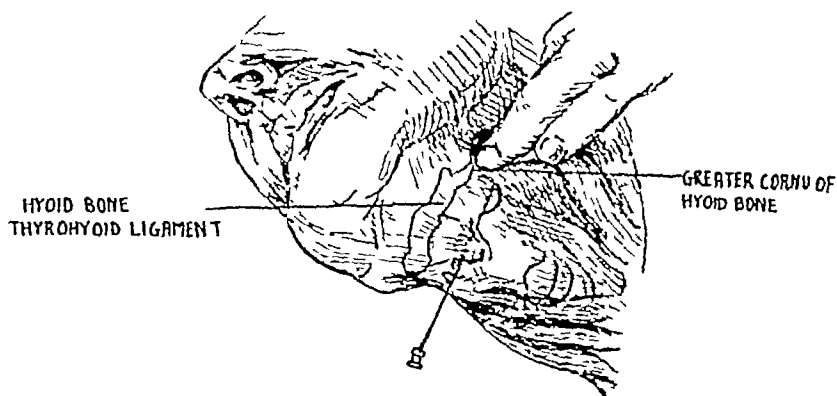


Fig 119—Injecting the superior laryngeal nerve

of the larynx and epiglottis, and relief from pain. Neurectomy of the internal branch gives the same results as alcohol injection.

#### RELIEF OF OCCIPITAL NEURALGIA

Occipital neuralgia is not common. When severe pain is experienced in back of the neck radiating over the occiput into the parietal region associated with hyperesthesia of the scalp, neuralgia of the great occipital nerve may be the cause but rheumatic myositis of the neck muscles, arthritis deformans, caries or tumors of the first or second cervical vertebra and hysteria must be ruled out.

The pain of occipital neuralgia will usually be relieved by external heat and counterirritants, as mustard plaster, to the occipital region, or alcohol injection into the great occipital nerve.

For alcohol injection, the needle is inserted vertically downward to the bone at the point of emergence of the nerve midway between the mastoid process and the first cervical vertebra, and about  $\frac{1}{2}$  inch below the level of the occipital protuberance. Subsequently there will be anesthesia of the scalp in the occipital region and relief from pain in that region.

## RELIEF OF PAIN FROM SLUDER'S NEURALGIA

Relief of pain from Sluder's neuralgia may be obtained by extirpation or cocaineization of the sphenopalatine ganglion

The treatment of this form of neuralgia is most efficiently performed by the nose specialist and the reader is referred to the literature on rhinology for further information on this subject



## CLINIC OF DR ROBERT O RITTER

### ST LUKE'S HOSPITAL

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#### RELIEF OF LUMBAGO AND SCIATICA

IN adult life there is no more common complaint than backache with or without sciatic radiation. The majority of patients with backache have, without a thorough examination, been treated for lumbago or sciatica. These two complaints and the disabilities resulting from them are serious problems which confront the general practitioner, the orthopedic and the industrial surgeon. Any patient seeking treatment for backache or sciatic pains requires a complete and thorough history and physical and x ray examination. Such an examination and history will, in most cases, lead to the discovery of the causative pathology. Only then can rational therapy be instituted.

There are many anatomic variations in the lower spine which may at times produce pain and disability. Among these are sacralized fifth lumbar transverse processes (Fig 120), spina bifida occulta, abnormal articulations, an abnormally oblique lumbosacral angle, spondylolisthesis (Figs 121, 122), pronounced hollow back and static factors. Flat feet and short Achilles tendons produce fatigue of the lumbar muscles, and often a severe back strain. Poor sitting posture in a chair or in an automobile during a long drive often causes backache.

Genito urinary, gynecological and neurological conditions should be investigated and eliminated. Pains in the lumbosacral region and over the sacrum should make one think of disease of the prostate and seminal vesicles. In women static backache often cannot be distinguished from that due to pelvic disorders. The gynecologist should be consulted in all ques



Fig 120

Fig 120—Mrs B I Sacralized fifth lumbar transverse process x-Ray showing tibial grafts in place



Fig 121

Fig 121—x-Ray of A S, showing fifth lumbar vertebra slipped forward into the pelvis



Fig 122—Complete fifth lumbar spondylolisthesis Two large tibial grafts were inserted across the lumbosacral joint.

tionable cases. A careful examination will eliminate many radical intrapelvic operations. However, it has been our experience that uterine trouble is more often ruled out than not.

Pain in the back is present in certain diseases of the nervous system. Tabes, cord tumors, disease of the cord itself, and peripheral nerve disturbances are to be ruled out. During the last year four patients, one man and three women, entered our clinic because of severe pain in the low back. Or



Fig 123

Fig 124

Fig 123—J P. Sciatic scoliosis with arthritis. Not relieved by palliative measures.

Fig 124—J P. After trisacral fusion. No return of symptoms after four years.

thopedic and x ray examinations failed to explain fully the cause for all the complaints. A thorough neurological examination revealed a multiple sclerosis in each case.

Metastatic disease of the spine and pelvic bones causes extreme pain. Pain due to malignancy is not relieved by rest on a rigid bed or by other therapeutic measures as is the pain from any other cause.

There can be no doubt that backache and sciatic pains are very often due to the presence of an actual osteo-arthritis



of toxic or infectious origin (Figs 123, 124) They may also be due to age and to certain back-straining occupations

When an inflammatory process is added to a mechanically unstable joint, pain and disability follow The pain may come on suddenly or insidiously, and may radiate down one or both sciatic nerves, the gluteal nerves or down the front or sides of the thighs It may be so severe as to be immediately disabling, as in sudden twists of the body or when a heavy object is lifted In many instances where there has been an injury, the pain does not appear until a day or two later The reason for this very common occurrence has not been fully explained By some it is considered a ligamentous strain followed by edematous changes or a tear of muscle fibers with resulting spasm By others it is regarded as a myofascitis of a rheumatic nature If the x-ray shows arthritic changes they are due either to disease or long continued strain or both

Many cases of disease and strain of the sacro-iliac joints occur but the lumbosacral is more often the site of the lesion In some cases it is difficult or impossible to determine in which of these joints the trouble exists However, a careful physical and x-ray examination will usually lead to a good working diagnosis

Pain along the course of the sciatic nerve occurs in both sacro-iliac and lumbosacral lesions If it is unilateral, either may be involved If bilateral, the lesion is usually in the lumbosacral joint Gaenslen's sign for sacro-iliac lesions is very reliable "Hyperextension of one hip with the opposite knee and hip acutely flexed to fix the lumbar spine, including the lumbosacral joint Pain is present only in the sacro-iliacs, ruling out lumbosacral cases"

The etiology of any given case will govern the treatment This may be either conservative or operative

All acute painful cases with or without radiating pains should be put at rest on a rigid bed, and some form of physical therapy, such as heat and massage, begun Large doses of salicylates are beneficial Head and pelvic traction or a plaster cast may be necessary to relieve muscle spasm in a

certain number of cases All foci of infection should be carefully sought and removed This is especially true in the intestinal tract, teeth and tonsils However, teeth should not be extracted just because they are devitalized (Fig 125)

In cases of low backache, where mild static faults are present, the skeletal alignment is corrected with supporting corsets or braces Graduated exercises, massage and physical therapy are of value In sacro-iliac strains without extreme

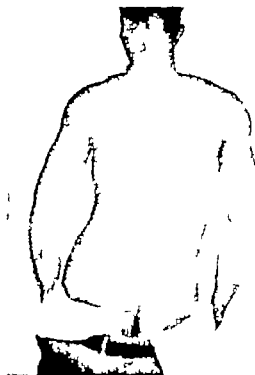


Fig. 125

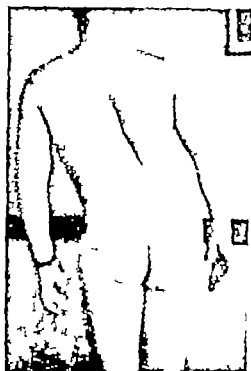


Fig 126

Fig 125.—J C Sciatic scoliosis Relieved by clearing up infected teeth and gums, and palliative measures.

Fig 126 —S K. L. Scoliosis. Severe lumbago Relieved by palliative measures

pain or muscle spasm, a firm adhesive plaster strapping gives immediate relief The one disadvantage of adhesive is that some skins are easily irritated by it For this reason a well fitting, non elastic corset is preferable In a great many cases of painful sacro-iliacs an ordinary belt such as men wear, buckled tightly around the pelvis just above the trochanters, gives prompt relief (Fig 126)

In some cases where the disability comes on suddenly following an injury, manipulation under complete anesthesia and

rest on a rigid bed for a few days are sufficient. Other more severe cases require immobilization with a cast or brace.

In the many cases not relieved by conservative measures, surgery becomes necessary. These are the chronic cases in which there is definite x-ray evidence of disease or deformity. In a certain number of cases, affection of the lumbosacral or sacro-iliacs cannot be differentiated. In these, all three joints should be ankylosed. For the lumbosacral arthrodesis the Hibbs technic has been quite generally used with the addition of two large tibial grafts. These grafts are cut from the flat surface of the tibia. One end of each is cut on an angle that just fits the lumbosacral angle. The grafts are fitted in edgewise, one on either side of the denuded spinous processes, with the medullary surfaces together. In all cases where this type of graft has been used, a very heavy mass of bone has been formed, and no failure of ankylosis has occurred. Such has not always been true where pieces of ilium were used.

The Smith-Petersen operation is adaptable to one or both sacro-iliacs, but not to the combined or triple arthrodesis. For the triple arthrodesis the extra-articular operation on the sacro-iliacs is the method of choice.

Operation is certainly advisable if the disability is a real handicap, especially in a wage earner. The operation is as safe as any major surgery, but it has a disadvantage in that it requires eight to twelve weeks for a solid ankylosis to take place.

When the excellent results from operative, as compared to nonoperative, treatment are compared, we feel that surgery is more conservative and time saving for the patient than mechanical treatment.

## CLINIC OF DR. LOUIS T CURRY

### COOK COUNTY HOSPITAL

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#### TREATMENT OF EARACHE

EARACHE (otodynia, otalgia) is caused by pathology in the middle ear, the external ear or the internal ear, or is referred from neighboring structures in the mouth, pharynx, larynx or nose. Earache is characterized clinically by pain in the ear region and pathologically by stimulation of the complicated sensory nerves of the ear mechanism.

As a foundation for this discussion, I shall first briefly summarize the sensory innervation of the ear mechanism, as taken from the anatomies of Cunningham and Spalteholz, and then proceed with a consideration of earache, its differential diagnosis and treatment.

##### *Sensory innervation of the ear mechanism*

##### I In the ear itself

##### (A) External ear

##### 1 Auricle

##### (a) External surface

- (1) Auriculotemporal nerve, from mandibular branch of fifth nerve supplies upper half
- (2) Great auricular nerve from C 2 to C 3 of cervical plexus, supplies lower half

##### (b) Cranial surface

- (1) Lesser occipital nerve from C 2 to C-3 supplies upper half
- (2) Great auricular nerve from C 2 to C 3 supplies lower half

##### 2 Scalp behind ear—lesser occipital and great auricular nerves

##### 3 Zygomatic region—auriculotemporal nerve from mandibular fifth

##### 4 External auditory canal and drum membrane

- (a) Auriculotemporal nerve from mandibular division fifth

supplies external auditory canal and external surface of the ear drum

- (b) Auricular branch from close below ganglion jugulare of vagus (Arnold's nerve) supplies concave surface of auricula and external acoustic meatus, and communicates with glossopharyngeal nerve

(B) Middle ear

- 1 Tympanic plexus supplies mucous membrane of tympanum and mastoid cells. Tympanic plexus is formed by tympanic nerve (from petrous ganglion of glossopharyngeus nerve) which enters the tympanum where it is joined by carotico-tympanic branches from the carotid sympathetic plexus on the internal carotid artery and a twig from the genicular ganglion of the facial nerve

(C) Inner ear

- 1 Supplied by two divisions of the specialized auditory nerve—cochlear and vestibular

II Neighboring structures

- (A) Otic ganglion. Fibers of tympanic branch of glossopharyngeal nerve reunite with small nerve from genicular ganglion of facial nerve (ramus anastomaticus cum plexus tympanico) to form the lesser superficial petrosal nerve. This nerve eventually ends in the otic ganglion on the trigeminus nerve

- (B) Sphenopalatine ganglion. Greater superficial petrosal nerve (mostly sensory fibers) from genicular ganglion of facial nerve unites with deep petrosal nerve from sympathetic on internal carotid artery to form nerve of pterygoid canal (O. T. vidian nerve). It passes through sphenopalatine ganglion and supplies mucous membrane of soft palate

- (C) Pharyngeal plexus. Pharyngeal branches of glossopharyngeal nerve supply mucous membrane of pharynx. These combine with pharyngeal branches from ganglion nodosum of the vagus nerve and the superior cervical ganglion of the sympathetic to form the pharyngeal plexus

(D) Tongue and tonsils

- 1 Lingual nerve from mandibular branch of fifth nerve supplies the anterior two thirds of the tongue, the mucous membrane of the side wall and the floor of the mouth. The chorda tympani from the facial nerve is carried in the lingual nerve
- 2 Terminal branches of the small glossopharyngeal nerve supply the posterior third of the tongue and the tonsils
- 3 Vagus nerve sends fibers to the base of the tongue

(I) Larynx and epiglottis

- 1 Glossopharyngeal nerve supplies anterior surface of epiglottis
- 2 Superior laryngeal nerve (internal branch from vagus) supplies mucous membrane of larynx and passes upward to epiglottis and base of the tongue

From this sketchy summary of the complex ear innervation one concludes that earache may arise from stimulation of C-2 and C 3 of the cervical plexus and cranial nerves V, VII, VIII, IX and X.

*Earache Its differential diagnosis and treatment*

I Aural pain from disease in the ear mechanism

(A) Middle ear disease causing pain

- (1) Acute catarrhal otitis media
- (2) Acute serous otitis media.
- (3) Acute suppurative otitis media
- (4) Acute mastoiditis
- (5) Chronic suppurative otitis media and chronic mastoiditis
- (6) Chronic adhesive middle ear catarrh
- (7) Blue ear drum

**1 Acute Catarrhal Otitis Media**—As a rule acute catarrhal otitis media is a complication of acute coryza. The patient notices a fulness in the ear which may later become severe. There may be crackling and buzzing noises in the affected ear. The ear drum usually shows some congestion of the blood vessels especially over the handle of the malleus. There is usually some diminution of hearing. The treatment consists in the treatment of the cause.

**2 Acute Serous Otitis Media.**—Acute serous otitis media arises when acute catarrhal otitis media progresses with the formation of serum in the tympanum. If the tympanum fills, the ear drum appears unusually clear, although the patient complains of fulness in the ear and the hearing is impaired. If the serum partially fills the tympanum, its upper edge may be seen as a hair line shining through the drum head. The pain is in the ear and usually disappears in a day or two. Treat the underlying cause. Four or 5 drops of warm 5 per cent carboglycerin may be instilled in the aching ear every two or three hours for relief of the pain. Application of heat is usually comforting. Hot dressings, hot water bag, electric pad or the incandescent light may be used. If the serum is not absorbed in a few days it is advisable to perform a myringotomy with careful antiseptic precautions. The serum is then blown out of the tympanum by inflation through the

eustachian tube Inflations may be done in three ways (1) Valsalva method—patient holds his nostrils, closes his mouth and employs forcible expiration (2) Politzer bag—blast of air is shot into one nostril while closing the other at the time patient is asked to say K or swallow a sip of water, (3) eustachian catheter—catheter passed through nose below inferior turbinate and inserted into eustachian opening Through catheter blast of air is applied, blowing secretion from tympanum Auscultation tube from affected ear to ear of operator is necessary to hear when this is accomplished

**3 Acute Suppurative Otitis Media**—Acute suppurative otitis media is the most frequent cause of earache Actually this condition is severe, acute, advanced catarrhal otitis media Therefore, it usually follows head colds or one of the exanthemata The pain is severe and is felt in the ear It is usually continuous but may have exacerbations It is usually worse at night The pain is relieved, as a rule, if the ear drum ruptures or if a paracentesis is done With severe infections, the earache may continue for several days even after drainage has been given The discharge may be serous to purulent with any degree of intermixture It may be slight or profuse Before rupture or opening of the ear drum, the drum head may be generally red to very red and bulging Blood vesicles may cover the ear drum and fill a large portion of the external auditory canal There is usually an elevation of temperature and a decided defect in the hearing

*Treatment* —1 External heat applied to the ear is the best method of relieving the earache This may be applied by means of a hot water bag, moist hot dressings or an electric pad Opium for relief of earache is contraindicated because it may mask the progress of the disease

2 Ear drops for relief of pain are prescribed by many otologists The favored prescription is phenol and glycerin (1 20) This mixture is to be used warm in the aching ear every two or three hours (3–5 drops)

3 *Myringotomy*—The ear drum must be opened as soon as any bulging of the drum membrane is seen or sooner if

the earache continues for twenty-four hours or longer, even in the absence of bulging. Mastoiditis and intracranial complications occur less frequently where early paracentesis is done. Paracentesis of the ear drum is carried out, using proper antiseptic precautions, with an anesthetic, either local or general.

*Local Anesthetic*—Equal parts of phenol, menthol and cocaine hydrochloride are used. This thin paste is applied sparingly with a small cotton applicator to the bulging area of the ear drum. It is well to warn the patient that the first touch hurts. The swab is held in place as long as the patient can bear it, which is usually a few seconds. The same procedure is continued until the applicator can contact the inflamed ear drum without much discomfort for a minute or two. The incision is then made through the bulging area of the ear drum, which should appear white if properly anesthetized with a light paracentesis knife. If there is no bulging the incision is made in the posterior quadrant of the ear drum. In certain types of influenza ears, the ear drum cannot be seen because of blebs. These can be opened only by guess. With gentleness and care, an experienced surgeon can insert the knife through the blebs and toward the ear drum until the point of the knife gently strikes the promontory without harm. I have found no need for extensive incision of the ear drum. If the tympanum continues to fill with pus, the small incision enlarges with the infection. If drainage is slight, the small or large incision closes quickly and another opening may be required.

*General Anesthetic*—Nitrous oxide or ethylene are ideal anesthetics for myringotomy. The advantages are evident, but there are drawbacks. The cost is greater for a general anesthetic, since more time is required of the physician, an anesthetist's services are required, and there is a hospital bill. Another handicap to the general anesthetic is that the indications for paracentesis often arise during the acute exanthemata while the patient is quarantined.



*Without Anesthesia*—Myringotomy may be performed without anesthesia, but the operation causes severe pain.

Larache may disappear after drainage has been given, to reappear if any blockage takes place. This can be remedied by instilling a few drops of hydrogen peroxide into the meatus. Thus drainage is reestablished. When earache continues after paracentesis, external heat is applied.

The draining ear should not be plugged with cotton. The external canal must be kept clean by dry wicks, frequently changed, or by frequent wiping of the external meatus. When the discharge is very thick, it may be cleaned out by gentle syringing with warm boric or soda water. At night, the ear toilet is best cared for by layers of gauze over the pinna which are held in place by a hair net tied under the chin. The cavum conchae and incisura intertragica must be kept clean with warm saline solution bathing daily and covered with a thick layer of zinc oxide ointment. If this is not done, the patient often develops a painful dermatitis from the irritating discharge.

**4 Acute Mastoiditis**—Acute mastoiditis is usually a complication of acute suppurative otitis media. Pain over the mastoid, often present when myringotomy is done, usually disappears after drainage is instituted. Pain over the mastoid, significant of acute mastoiditis, usually starts after the ear has been draining for two or three weeks. It may, however, be present in the beginning of the otitis media and persist until relieved by surgical intervention. In other cases, it may appear after the ear discharge has ceased. There may be changes in the soft parts over the mastoid process and tenderness to pressure, especially over the antrum, the tip or the emissary vein. In adults there may be no tenderness when pressure is applied over the mastoid or alterations in the soft structures even in the presence of an acute mastoiditis.

Mastoid pain is usually worse at night. If the patient with an acute otitis media has pain at night in the mastoid area and a discharge which is becoming more purulent, formation of a mastoid abscess is indicated. Pain may also be

caused by subperiosteal abscess or Bezold's abscess. The pain is less after the abscess forms than during its formation. Tenderness over the abscess is noted upon palpation. The subperiosteal abscess may form posterior, superior or anterior to the auricle. A Bezold abscess is caused by a rupture of a mastoid abscess through its tip. These may appear superior, in or below the sternocleidomastoid muscle. Occasionally they appear posterior or anterior to this muscle.

*Treatment*—Early mastoid pain is treated by hot applications as previously described. Some otologists prefer ice packs or ice coils, feeling that an abscess is more often avoided. The cold aids in control of the pain.

The indications for operative interference in acute mastoiditis differ for the small child and for the adult.

In a small child operative interference is indicated.

1. When subperiosteal abscess develops if the ear has previously had proper treatment and drainage.

2. If symptoms of threatened intracranial complications appear, as meningismus, meningitis, lateral sinus thrombosis, brain or cerebellar abscess.

In an older child or in an adult the indications for operation are:

1. As above.

2. As above.

3. If earache continues for seven to ten days after proper treatment has been instituted.

4. If the discharge continues to be purulent after proper treatment and shows no evidence of diminishing for four weeks.

5. If the patient continues to have rather high fever and palpable changes over the mastoid for two weeks, even though proper treatment has been given.

The operation indicated is a simple mastoidectomy which has for its object the drainage of the mastoid abscess. Usually in adults the external wall of the antrum is opened, although the antrum is not necessarily the location of the abscess. In

infants, since mastoid cells have not developed, the operation is more properly called an antrotomy

**Recurrent Mastoiditis** —This term is applied to patients who have had simple mastoidectomies and develop acute suppurative otitis media. The pus backs up into the antrum which is immediately below the soft parts behind the auricle. An abscess may develop in this region which causes the pain typical of any abscess. After using heat, if the abscess does not subside, it is opened and drained as any other abscess, after which the symptoms of otitis media usually promptly subside if the previous mastoidectomy has been complete. Occasionally it is better to expose the mastoid cells area and seek for further infected broken-down cells which should be eradicated.

**Gradenigo Syndrome** —This properly comes under the heading of mastoiditis. Severe headache referred to the side of the head in the region of the affected ear, purulent otitis media and paralysis of the sixth cranial nerve on the same side are the cardinal symptoms of this syndrome.

**Treatment** Most otologists agree that at least a simple mastoidectomy is indicated when symptoms of this syndrome are present. Others insist upon drainage of the petrous tip cells, which is a difficult and dangerous procedure. Swelling of the gasserian ganglion which causes the sixth nerve to be put on a stretch is one of the theories given for paralysis of this nerve. In these conditions I favor conservative treatment as taught me by Dr. George E. Shambaugh. We used conservative treatment on a woman with chronic suppurative otitis media accompanied by Gradenigo's syndrome five years ago. She recovered and has been free from recurrence since. I have treated twenty patients successfully by conservative treatment, *i. e.*, simple mastoidectomy and keeping the wound open until dry. One of my patients died. His pain continued in the side of the head after the mastoid operation. Drainage continued for one month, after which the pain subsided and the drainage ceased. He moved to another city, had a recurrence of headache, was operated and died.

I do not know the nature of the operation performed. Recently I treated a boy with typical symptoms of this syndrome, but his otitis media was mild. His aural discharge stopped, his headache subsided and his lateral rectus paralysis disappeared in two weeks. I decided not to operate upon his mastoid. His ear is now dry, his drum membrane healed and he is free from headache but his lateral rectus paralysis has reappeared. He has been under observation for three weeks since his ear drum became normal. The outcome is to be awaited.

5 Chronic suppurative otitis media and chronic mastoiditis are not as important from the standpoint of earache as they are from absence of earache and danger of intracranial complications. Chronic suppurative otitis media may be discussed under two very different types.

1 Inflammatory changes limited to mucous membrane of the middle ear chambers. This is misnamed the "nondangerous type," but should be called the less dangerous type.

2 Changes in the middle ear associated with a bone-invading process. This is properly called the dangerous type.

In the first type, pain is rarely a symptom if the ear is kept clean and properly treated. Occasionally a polyp forms which blocks drainage and causes earache. The treatment is to remove the polyp with an ear snare or cautery and reestablish proper drainage. Silver nitrate in solution or fused on a probe, or chromic acid fused on a probe may be used to cauterize. In using the chromic acid bead, the bead should be cherry red and not black in color to cauterize properly.

Individuals with chronic otitis media sometimes develop an acute otitis media in the same ear which gives the otalgia of that disease. The treatment is practically the same except that operative interference may be indicated earlier because of the greater damage of intracranial complications.

In the second type of chronic suppurative otitis media, earache is more common but seldom severe. It consists usually of a dull ache in the ear or is referred to the side of the head as a heavy feeling. Occasionally the pain may be deep

seated or boring in character. Its significance is quite important, since it may indicate a threatened intracranial invasion. In this type of ear disease we deal with sequestration, caries and cholesteatoma. Usually the treatment leads to radical operative treatment, unless the hearing in the opposite ear is more seriously affected. The radical mastoid operation is practically never done because of pain. Therefore, a description of it is not in order here.

The complications of chronic suppurative otitis media should be mentioned, but earache is not a prominent sign in any of them. They may intervene without any sign of earache or there may be a warning sign of a heavy feeling in the side of the head. Very rarely is there any severe pain.

#### INTRACRANIAL COMPLICATIONS OF CHRONIC SUPPURATIVE OTITIS MEDIA

1 Labyrinthitis, which may be circumscribed or diffuse and serous or suppurative

2 Lateral sinus thrombosis

3 Meningitis, which may be serous or suppurative and circumscribed or diffuse

4 Brain abscess

5 Cerebellar abscess

6 **Chronic adhesive middle ear catarrh** occasionally causes dull or even sharp earache. Prophylactic treatment of this condition is most important, because if neglected it causes permanent impairment of hearing. Removal of tonsils and adenoids early, in children suffering from repeated ear infection, is the best prophylaxis.

7 **Blue ear drum** is a rare condition in which the tympanum is filled with a thick chocolate-like secretion. Inspection shows the blue ear drum. It causes more of a fulness than an earache. Treatment is the same as for serous otitis media.

#### (B) External ear disease causing pain

##### 1 Inflammations

##### (1) Furunculosis

- (b) Eczema
- (c) Fungi
- (d) Erysipelas
- (e) Subperichondrial abscess
- (f) Herpes zoster auriculæ
- 2 Foreign bodies
  - (a) Inanimate
    - (1) Impacted cerumen
    - (2) Vegetable as peas corn beans
    - (3) Miscellaneous—stones, paper pencils etc.
    - (4) Epithelial plugs
  - (b) Animate
    - (1) Insects
    - (2) Worms
- 3 Trauma
  - (a) Burns
  - (b) Frostbite
  - (c) Pugilist ear
  - (d) Wounds—gunshot stab etc
  - (e) Rupture of ear drum penetrating wound blow skull fracture
- 4 New growths—carcinoma
- 5 Granulomata
  - (a) Syphilis
  - (b) Tuberculosis
  - (c) Blastomycosis
  - (d) Actinomycosis

(a) **Furunculosis** of the external meatus is always located in the outer membranous portion of the meatus and the diagnosis can nearly always be made by eliciting pain by pressure upon the meatus or by movement of the auricle. If the furuncle is deeper in the meatus and large, it may push the auricle forward, simulating a subperiosteal abscess in acute mastoiditis. Both conditions may be present. Often furunculosis is associated with an acute or chronic suppurative otitis media. In these instances the signs of inflammation as given above are found in the external portion of the meatus and the indications of the middle ear disease are discovered with speculum and by means of tuning fork examination.

The pain in furunculosis is often severe and out of all proportion to the clinical findings. It is referred to the ear and the side of the head, and often mastication is painful.

*Treatment*—Prophylaxis “Avoid scratching the ears with anything but the elbows” is trite but excellent advice. For itching ears use salicylic acid in alcohol (1-10 per cent). Swimmers may avoid furuncles if boric acid and 95 per cent alcohol (4-100) is used to dry the meat after coming out of the water.

The active treatment of furunculosis should always be conservative. My preference is to carefully wipe out the meatus and fill with  $\frac{1}{2}$  of 1 per cent yellow oxide of mercury in water-free lanolin. This procedure should be repeated daily by the physician until the furuncle ruptures or subsides. At home the patient should use wet hot dressings of generous size over the auricle constantly if the pain is severe. Sedatives may be prescribed, as acetylsalicylic acid, acetanilid, salicylates, even morphine. With this treatment the patient suffers usually two or three days, but recurrences are rare. In infants the same treatment may be used. Phages and anti-virus may be used. Five per cent carbolic glycerin, 4 or 5 drops every three or four hours, may be tried in the ear early. When the furuncle has developed to a distinct white head, it may be incised with a cataract knife, using proper antiseptic precautions. Roentgen rays may be tried when the furuncle does not point and great induration is present. The dosage is usually about one-third erythema dose. Wick treatment is used by some otologists. Moist wicks are used, saturated with a weak solution of aluminum acetate or other astringent drug. Dry treatment is preferred by some. This may be carried out by blowing boric acid powder into the meat.

For recurring furuncles, good results are often obtained with autogenous vaccines.

(b) **Eczema** of the auricle and external meatus may occur in any form, but does not produce earache unless complicated by furunculosis or perichondritis. Itching is the chief symptom. The treatment (rather unsatisfactory) is both general and local, but does not fall properly under the title of this paper.

(c) **Fungi (Otomycosis)**—A soft, dirty, grayish, salve-

like substance may form in the external meatus. When this is removed, the canal wall appears raw and bleeds easily. This material recurs in a few weeks after cleaning if the canal is not treated. Subjectively the patient complains of a fulness, stinging or itching in the ear. The infection may be resistant to treatment. Usually a mild parasiticide is prescribed, as 2 to 10 per cent salicylic acid in 95 per cent alcohol. The ear is wiped out fairly dry and the patient instructed to fill the canal once or twice daily with the mixture. Hyposulphite of soda (0.2 in 30) may be tried. In a few days the fungus growth may be destroyed, after which it is well to have the treatment continued at weekly intervals for some time to prevent recurrence.

(d) **Erysipelas** of the auricle does not differ from erysipelas in other parts of the body. The pain may be intense in the involved region and the disease presents its redness, swelling, raised, red border and fever.

*Treatment*—Prophylaxis and isolation from surgical and obstetric patients until lesions are healed. Locally, the treatment is ultraviolet irradiation of the area involved and of the normal adjacent skin at least 2 inches beyond the border. Roentgen rays are used not over one-fourth erythema dose. Evaporating lotions are applied ice cold by means of uncovered compresses. A 50 per cent solution magnesium sulphate may be used. Erysipelas antiserum preceded by an intracutaneous test of 0.1 cc. of the serum diluted 1:10 may be used. If the test is negative within fifteen minutes, the remainder of the contents of the ampule may be injected. The dose may be repeated daily for three days. If the test is positive, it might be well to try convalescent erysipelas serum rather than the serum derived from the animal, in amounts varying from 40 to 100 cc. Fever symptoms are treated as indications arise. Hypnotics, often paraldehyde (teaspoonful in a half glass of water), are used for obstinate insomnia, and stimulants for collapse.

(e) **Auricular perichondritis or subperichondrial abscess** is ushered in with pain over the auricle affected, the



pain depending on the severity of the infection and the area involved. The treatment depends on the extent of the lesion. If a relatively small area is involved, conservative treatment with wet dressings is indicated. If it is more extensive, it is best to incise early and drain with a small gutta-percha drain. Warm dressings are applied and the drain changed daily. The bandage should be rather tight. The principle is to save as much cartilage as possible because of the rapid cartilage necrosis and the consequent disfiguring deformity.

(f) **Herpes Zoster Auriculæ (Koerner's Syndrome· Hunt's Syndrome)**—Herpetic eruption over the concave surface of the auricula and the external acoustic meatus may be associated with facial paralysis. The pain is burning in character. Since the pathology is thought to be a geniculate ganglion neuritis, the treatment is that of any neuritis. Remove foci and give salicylates. One to 5 per cent ammoniated mercury ointment may be tried locally.

**Foreign Bodies**—Impacted cerumen is the most common foreign body but causes the least pain. If there is any pain, it is usually a fulness in the affected ear or occasionally an unpleasant sensation of something striking the ear drum with a change of position. Treatment consists in removal by syringing with an ear syringe, using warm water. The stream should be directed toward the upper portion of the canal while the auricle is pulled upward and backward. If several attempts prove unsuccessful, the patient is instructed to use normal salt or soda water in the ear twice daily and to return for a second syringing. The ear hook may be used if the operator is careful and skilful enough not to injure the delicate epithelium of the external meatus. The hook is placed over the cerumen, turned, and gentle traction is exerted. The cerumen, if dry and hard, is adherent to the dead surface epithelium. Therefore, the patient should be cautioned to hold still, and to swear, not jump, if hurt.

Vegetable foreign bodies, as peas, corn and beans, may swell after lodging in the external meatus and thus cause con-

siderable pain They can usually be syringed out if found early Otherwise they must be removed with a hook or ear forceps Great care must be exerted to do no harm in treating foreign bodies It is far better to leave any foreign body in the meatus than to injure the epithelial lining or push the object through the ear drum while trying to extract it.

Stones, paper and muscellaneous materials are often found in the external ear and may or may not cause earache They are removed as described *Hard foreign bodies*, when medial to the isthmus of the meatus, may be difficult or impossible to dislodge A modified radical mastoid operation may be necessary A considerable number of serious complications are seen each year because of clumsy attempts at removal of foreign bodies

Epithelial plugs (cholesteatoma like accumulations) are rare in the external auditory meatus, but may cause earache and impaired hearing if present The treatment is to remove the mass by mechanical means or by a solution which is dehydrating, as 1 per cent salicylic acid in alcohol After the canal is cleaned, the granulations should be treated until healed

Live insects may be very painful as they hop up and down on the ear drum A few drops of alcohol or chloroform quiets them, after which they are easily syringed out. In emergency, pouring water into the meatus will subdue these insects

**Trauma**—Burns demand no special consideration, as they are treated the same as burns elsewhere

Frostbite frequently affects the auricles because of their exposed location Instead of painful ears, the ears feel numb They should be rubbed with snow or cold hands This treatment restores the circulation When the ears become painful they are treated as burns

A prize fighter ear is usually caused by a blow on the auricle, or by a twist Pain depends upon the amount of blood accumulated beneath the perichondrium, but is usually not severe If the effusion is small, it should be treated conservatively and will absorb with a pressure bandage If the

blood clot is larger, free incision through the perichondrium is necessary and a pack is inserted as in perichondritis.

Wounds need no special description.

Traumatic rupture of the ear drum by puncture wound, a blow on the ear, explosions, or skull fracture may be accompanied by severe pain in the ear. Treatment is to control the hemorrhage, if necessary, by sterile tampon. In most cases all that is necessary is to prevent infection to the middle ear by using sterile cotton in the external meatus. Treat middle ear suppuration if it occurs.

**New Growths**—Epithelioma may attack the auricle or meatus. Pain is not a prominent feature. The treatment does not vary from the treatment of carcinoma elsewhere. Early excision or diathermy is indicated. If seen later, x-ray and radium may be tried. If the temporal bone becomes involved, the earache is one of the most terrible of pains and the treatment is unsatisfactory.

Endothelioma occurs in the ear usually secondary to parotid involvement.

Sarcomas, lipomas and neuromas are rare.

**Granulomata**—Syphilis and tuberculosis may occur on the auricle, but pain is usually insignificant. Blastomycosis and actinomycosis may involve the ear and cause moderate earache. A biopsy is usually needed for the diagnosis. I recently diathermized a blastomycosis of the lobule of an ear which I felt sure was epithelioma without waiting for a biopsy report. I should have suspected blastomycosis because of the large amount of destruction in nine months' time. Iodides in massive doses daily for a long time are the sheet anchor of treatment of these mycoses.

(C) **Internal Ear Disease Causing Earache**—Involvement of the labyrinth and brain due to middle ear disease has been discussed. There is no earache as a rule from internal ear disease caused primarily from meningitis, scarlet fever, mumps, measles, influenza or typhoid fever. There is only tinnitus.

## II Reflex Aural Pain.

*Toothache* frequently causes referred pain to the ear. The wisdom teeth are the chief offenders, the lowers more often than the uppers. Impacted molars which are causing otalgia may be discovered by a skiagraph. The treatment is the extraction of the offending tooth or teeth.

*Acute tonsillitis*, peritonsillar abscess and post tonsillectomy infection may give rise to earache, but usually there is no difficulty in the differential diagnosis and the treatment is self evident.

*In parotitis* the pain may be referred to the ear, but diagnosis is simple unless the external meatus is encroached upon so much that the ear drum cannot be examined.

*Laryngitis* caused by tuberculosis, cancer or other granuloma may cause otalgia. In these cases there is usually hoarseness and when the larynx is examined, the lesion is found. Biopsy is often necessary to make the differential diagnosis. Disease of the larynx causes earache by irritation of the superior laryngeal nerve, a branch of the vagus. The treatment is self-evident but one point might be mentioned. In dysphagia the superior laryngeal nerve is sometimes isolated and severed. When isolated, it is pinched gently, which causes pain in the ear and proves that the proper nerve is about to be cut. This symptom is also used as a guide for nerve block in dysphagia.

*New growths* in the vault of the pharynx and in the retropharyngeal region often cause earache. This pain may come from pressure on the nerves in this region or by pressure on the eustachian tube orifice, causing middle ear disease. These tumors are treated surgically or with x ray and radium.

Other conditions causing neuralgic pain in the ear are carcinoma of the superior maxilla, cancer of the tongue (posterior third), inflammation of the gasserian ganglion, cold air, anemia, hysteria and neurasthenia. Treatment consists in treating the etiological factor.



## CLINIC OF DR CHARLES W FREEMAN

### NORTHWESTERN UNIVERSITY DENTAL SCHOOL

#### PAIN IN THE DENTAL FIELD

PAIN in the dental field is usually definite in character and location, and in most cases the cause can be determined and readily eliminated by the available methods of diagnosis and treatment. Occasionally certain atypical and migratory pains require careful study for accurate determination of the cause and not infrequently search must be made outside the oral cavity.

Toothache is not in any sense a modern disease nor has its incidence been greatly reduced in modern times. It is however, a preventable disease or rather it is a symptom of a pathological disturbance which to a great extent is preventable. Dental caries is probably the most common of all diseases and at the present time is not entirely preventable, but it can be controlled. Modern dental care which includes periodic examination of the mouth and teeth with proper control of caries by fillings should prevent entirely the development of toothache.

#### PULPITIS

Pain known as toothache is caused by an hyperemia of the dental pulp, that highly innervated soft tissue which is entirely enclosed within a pulp canal and surrounded by dentine. Its blood supply is by way of one or several small foramina at the apex of the tooth, a fact which precludes the establishment of collateral circulation, and consequently tends to complete infarction when the pulp is seriously disturbed.

When an inflammatory process develops as a result of irritation—traumatic, chemical, thermal or bacteriologic—the en-

closing dentine permits no expansion of the inflamed tissue and a sharp pain ensues. Such a pain may be steady or intermittent sharp, dull or throbbing, depending on the local factors. It may be stimulated by a single sudden irritation, beginning as a sharp stabbing pain which gradually recedes. It may be aggravated by recurrent irritations, as in thermal shock where large metallic fillings are close to the pulp, the transmission of all extreme changes in mouth temperature repeat the insult to the pulp, thus further increasing the inflammation.

A steady pain of the same general type but without recurrent external cause generally indicates a partial degeneration or necrosis of the pulp. Infection may be present or absent within the pulp at this stage of degeneration. This pain occurs most frequently at night and there is at least a partial remission during the day, although the pain may be stimulated by outside irritations. Usually, in fact, such a tooth is extremely responsive to external stimuli and the patient is inclined to favor and protect it by chewing on the opposite side and avoiding extremes of temperature within the mouth. At night the pain is likely to be throbbing and the pulse can be distinguished by waves of increased pain.

Diagnosis of the cause of these pains is made by clinical study, augmented by thermal and electric tests of the suspected pulps. Often the patient can identify the offending tooth and clinical examination reveals an all too evident cause. The x-ray is not often useful, since pulp changes are not demonstrated on the x-ray film, although in pulpitis of long standing periapical bone changes may occur sufficiently to be recorded on the film while the pulp is still vital. The x-ray film is also useful in discovering hidden causes such as leaking fillings, undiscovered caries and, of course, is useful in ruling out other possible sources.

Occasionally the location of the pain is not definite and if an evident cause such as open caries is not discovered, it requires a careful diagnostic study to determine the source. Generally a delay of a day or two will permit further progress.

of the pulp degeneration with consequent definite localization of the pain and at that time any doubt as to location and cause is removed

The causative factor in pulp irritations may be the formation of secondary dentine such as occurs in connection with severe abrasion of the teeth. This secondary calcification may be so extensive as to completely obliterate the pulp chamber, the pulp receding as the secondary calcification takes place. This is an emphasized physiological action and normally occurs without pulp irritation, but may occasionally be responsible for severe pulp disturbances accompanied by pain.

In any pulpitis regardless of the cause, the recovery of the pulp is doubtful if the pain has been of long duration. The treatment consists of the extraction of the offending tooth or the surgical removal of the pulp with subsequent treatment of the pulp canal and placement of a root filling.

#### PERIAPICAL INFECTION

Pulp degeneration with eventual death of the pulp by infarction may be a rapid process accompanied by mild or severe pain symptoms just discussed, or it may occur with little or no pain and may be a very gradual process extending over months. Ultimately the death of the pulp does occur and a new type of pain may result. This is the pain connected with periapical inflammation and infection, and is of an entirely different character.

This pain may begin before the pulp degeneration is completed and this makes an accurate diagnosis difficult, because one type of pain is merging into the other, consequently the pain is not typical of either pathological condition. Usually the period of transition is not prolonged, since inflammation in the periapical region causes additional disturbance to the blood supply of the degenerating pulp and complete infarction rapidly follows. It should be reiterated that both these conditions may occur as a result of irritation without infection, but even if infection is not present in the early stages it almost invariably occurs later if treatment is not given.



Pain connected with periapical infection is distinguished from pain within the pulp by the character of the pain and the reaction to external stimuli. In periapical infection the pain is definitely one of pressure, it is generally well localized and a certain tooth is sore to percussion and pressure and the tooth gives a sense of elongation due to the pressure of confined fluids in the periapical space. At this period or a little later the gum tissues over the apex of the tooth also become inflamed and sore to pressure.

A controlled inflammatory process probably occurs in the periapical region in every case in which the pulp dies or is artificially removed. If infarction of the pulp occurs without bacterial invasion or if the pulp is removed surgically by dental methods, the disturbance of the periapical tissues and the consequent pains are slight and transitory. Occasionally chemical irritation with considerable pain occurs during the treatment of a tooth with certain drugs used for sterilizing the root canal. This also is transitory unless metastatic infection occurs.

The initial periapical bone changes which can be depicted on the x-ray film are probably caused by the inflammatory reaction with pressure resorption of the bone. When infection occurs bacterial liquefaction of the bone follows and a definite bone cavity develops. Coincidentally, an inflammatory proliferation of the connective tissue of the peridental membrane occurs, and this proliferated tissue eventually becomes the so-called "granuloma" of the chronic periapical infection. The granuloma is a protective rather than a destructive tissue and serves to wall off the infection, and under favorable conditions the infection may actually be eliminated with eventual regeneration of bone.

During the early stages of the acute periapical disturbances infection is not under control and the severe pain increases and continues for several days. Unless relief is given by treatment through the root canal the infection develops to a point where evacuation through the bone relieves the severe pressure. In the occasional case the infection is controlled

by the tissue response and spontaneous recovery from the acute phase of the infective process occurs

When the acute infection continues to the localization of pus there is usually a definite time at which the severe pain from confined infection is relieved by evacuation through the bone. The pain then becomes more generalized, but the tooth is no longer sore, for the tissues overlying the bone in the periapical region are now exhibiting the symptoms of acute infection. General toxic symptoms also increase at this stage but such symptoms seldom occur while the pulp is still vital regardless of the amount of pain which may be present.

After localization of pus beneath the periosteum the proper treatment is evacuation of the pus by incision or if untreated there is usually an eventual spontaneous evacuation. Any delay in drainage after localization is dangerous, for subperiosteal accumulation of pus seriously interferes with the blood supply to the bone and may result in serious bone disturbance.

Pericoronal infections about partially erupted teeth, and occasional lateral abscesses due to deep pyorrhea pockets alongside the root of the tooth may also exhibit some of the symptoms described in periapical infections. Generally the first phase of the infection with the typical soreness of the tooth to percussion is absent in such infections, since they do not begin in the apical region. They are also more likely to end in spontaneous drainage, since the initial infection is not located deep within the bone.

A large percentage of the pains in the oral cavity are of the types just discussed. Accurate diagnosis is usually possible at the first clinical examination. Occasionally observation for a day or two is necessary to determine the exact source of the pain, but since both types of disturbances are progressive in character any uncertainty may be eliminated by a short period of clinical observation. After the cause of a pulpitis or a periapical infection has been determined definite treatment is readily planned and practically always effective.

### MAXILLARY SINUS INFECTION

Maxillary sinus disease is occasionally of dental origin, but the initial cause is more frequently nasal. Pain in the dental field may be caused by sinus infection regardless of the original source, since the roots of the upper teeth are frequently in close proximity to the maxillary sinus. In addition to the common symptom of a general feeling of pressure in the sinus region there may be a definite sense of soreness of the teeth. This pain is very similar to a beginning periapical infection and may occasionally appear to be localized definitely in an upper bicuspid or molar tooth.

x-Ray studies may not always rule out the teeth, and pulp tests are only an indication, not an accurate diagnostic aid. Adequate sinus examination by a rhinologist should be obtained in cases of uncertainty. On the other hand, a periapical dental infection may simulate a maxillary sinus infection and in any uncertainty a careful dental examination is essential. Probably a careful study of all the upper teeth should be routine procedure in all maxillary sinus infections.

### UNERUPTED AND IMPACTED TEETH

Vague pains about the face and jaws are frequently attributed to the presence of embedded and unerupted teeth. No satisfactory explanation of the mechanism causing such pains has been advanced, but their removal is often recommended in the expectation of relief.

Unerupted teeth may become infected if there is direct communication with the mouth cavity and infrequently they become infected when no such communication exists. Such an infection runs a typical course and is often responsible for local pain, but it is the typical pain of local infection and is accompanied by clinical signs of infection. Pains which are intermittent in character or sharp shooting neuralgic pains are not caused by local infection about unerupted teeth.

Direct contact between an unerupted tooth and its immediate neighbor may cause a pressure resorption of the root of the contiguous tooth, thus setting up a severe pulp irrita-

tion with accompanying pain. Such resorptions can usually be identified by radiographic study and the usual symptoms of pulpitis are generally definite and well localized. Caries occurring in the partially erupted tooth or in its immediate neighbor as a result of food impaction may occasionally give the same symptoms of pulpitis. The cause is always demonstrable by radiographic and clinical examination when this does occur.

There is no valid reason to believe that the presence of an unerupted tooth in itself causes any pain, although some clinicians have expressed the belief that this does occur. It is explained on the basis of intermittent efforts on the part of the tooth to erupt, the eruptive force causing pain by pressure on adjoining structures. This hypothesis does not appear tenable, for pressure resorption occurs in advance of eruption and if bone structure is the only impediment to eruption, that bone resorption would be slow and painless. Innumerable clinical cases occur in which teeth, which are prevented from erupting, remain for years with no painful symptoms.

Vague pains and other nerve disturbances in the facial region which cannot be satisfactorily explained should not be attributed to unerupted teeth simply because they are found to be present. Careful examination may reveal the presence of caries, resorption of the contiguous tooth or pericoronal infection, but few, if any, other pains are caused by unerupted teeth. Follicular cysts occasionally develop about unerupted teeth, but they are painless unless infected. Supernumerary teeth and composite odontomes are frequently present about the jaws, but are not responsible for pain symptoms except under the same conditions which cause pain about unerupted teeth.

### NEURALGIA

Pains generally classified as neuralgia may be considered as major and minor neuralgia. Major trifacial neuralgia or tic douloureux exhibits typical symptoms and a definite diagnosis is practically always possible. Sphenopalatine neural

gia is also typical in its symptoms and although infrequently encountered the diagnosis can generally be made on the basis of symptoms

Minor neuralgia about the teeth and jaws is probably most often a pain symptom with a definite local cause which can be discovered by painstaking search and should not be classed as a neuralgia. Dental causes are then the most frequent sources of these pains in the regions innervated by the second and third divisions of the fifth nerve. These causes are frequently obscure and it requires a most careful survey of every possible source to determine the causative factor. This survey should include complete radiographic study of the jaws including the edentulous areas.

Such a study will include a search for dental caries, infections, retained roots, supernumerary and impacted teeth, imperfect and irritating dental restorations and any abnormality of bone structure. Disease of the maxillary sinus should be considered and ruled out as well as any nasal disturbance which might be a contributing factor.

Infrequently such pains may be caused by local nerve irritations in the region of a previous operation. Not only fractured retained root tips, but rough margins of alveolar process may be acting as local irritations. These occurrences are not common, for usually root fragments are exfoliated or encapsulated, the encapsulated fragments becoming surrounded by a low-grade chronic infection which does not cause pain. Rough margins of alveolar bone following extraction of teeth are generally resorbed or exfoliated and the tissues make the necessary adjustments without painful sequelae.

Several clinicians have reported the relief of such pains by minor operations which released small nerve filaments from the pressure of contracting scar tissue where operation had previously been performed, especially in the regions where impacted teeth or large cysts had been removed. This is a plausible explanation and possibly has some basis of fact, although such cases are so infrequent that there is considerable doubt of their actual significance.

## EXTRAORAL CAUSES

Among the extraoral causes for pain in this region intra cranial disturbances, especially brain tumors, must be considered as the most important. On several occasions I have been requested to remove impacted teeth for the relief of severe pain, when a more complete medical and neurological examination proved the source of pain to be a brain tumor.

Traumatic causes are usually self-evident and easily determined as the cause of pain. Tumors along the course of a nerve occasionally cause pain, but when this does occur it is a different type and not easily confused with pain of dental origin.

Arthritis of the temporomandibular joint is infrequent and the pain is definitely localized and is increased with functional movement. Pain which is due to changed relations of the structures of the temporomandibular joint, as a result of the loss of teeth or changes in occlusion due to wear, are usually referred to the region of the ear rather than the oral cavity.

## BURNING TONGUE

An affection of the nerves, described as burning tongue, occurs infrequently. It is not a pain, but rather an unpleasant sensation, quite constant but with remissions. I have seen this most frequently in women during the menopause and it may probably be attributable to derangement of the entire nervous mechanism rather than to any local source. It has been suggested that the explanation may be based on electrophysical reactions induced by the presence of metallic filling in the teeth. There has been no verification of this hypothesis.

## SUMMARY

The pains about the oral cavity may be grouped as

- 1 Irritations and infections of the dental pulp
- 2 Infections and inflammations at the root ends
- 3 Disturbances about unerupted teeth
- 4 Pains not of dental origin

The diagnosis of the cause of such pains is usually possible with the available clinical, radiographic and laboratory methods. Occasional severe pains occur in which the cause is obscure and continued search may fail to reveal any local cause. When such pains are of dental origin and the proper diagnosis is made, definite treatment may be adopted which will rapidly and effectively eliminate the pain.

## CLINIC OF DR. WALTER R. FISCHER

### ILLINOIS MASONIC HOSPITAL

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#### RELIEF FOR PAINFUL FEET

THE successful relief of pain in the feet requires as the first step the correct interpretation of the pain. A thorough knowledge of factors that produce pain in this part of the body is of the greatest importance for the analysis of each case presented. The need of a careful general survey of the patient cannot be too greatly emphasized. Pain in the foot may be the manifestation of some constitutional disease or the result of some local disturbance. One can distinguish only by a thorough physical examination. A systematic study should be made of the character, location, time of occurrence, duration and mode of onset of the pain together with a careful consideration of the associated factors such as systemic disease and local changes or abnormalities.

The things that must be taken into consideration in the history of every case are the age, sex, occupation, weight, the possibility of systemic disease, disturbances in posture and the elements presented by the feet themselves, along with the type of wearing apparel.

The cause of the pain varies to a great extent with the age of the individual. For instance, in the very young child one may find the cause to be a weak pronated foot due to faulty use or it may be associated with a deformity of the lower extremity such as genu valgum, knock knee, which disturbs the line of weight upon the foot. The older child or adolescent may suffer from foot strain brought on by the more



strenuous athletic games or the burden of occupation. Discomfort in middle-aged adults may result from obesity, arthritis or faulty shoes, while persons in advanced age may complain of trouble appearing as the manifestation of vascular disease. In other words, the age is more or less an index of the kind of trouble that is possible.

A very important distinguishing feature in the analysis of foot discomfort is the sex of the person. Women make up the great majority of individuals seeking relief and in a large percentage of the cases the modern style shoe is responsible for the production or aggravation of painful foot disturbances.

Occupation plays an important part in the production of pain in many ways. It may call for long hours of standing, which is usually done with the feet in a pronated position, thus weakening by exhaustion the upward pull of the adductor muscles of the foot. A patient's duty at work may require the lifting and carrying of heavy objects, walking over rough and irregular tracts of ground, or walking on hard pavements for many hours. A person who has escaped trouble for many years at a desk job may be transferred to a position that requires a great deal of activity upon the feet and as a result suffer great discomfort.

Since the functions of the foot are to support and move the body about, the influence of weight in every instance must be determined. Given a normally functioning foot with an extra heavy body superimposed, there is bound to be sooner or later a change in the comfort of the foot, and from no other cause than the additional weight burden. When the factor of excess weight has added to it the presence of a pathological condition in the foot such as for example arthritis, the symptoms of distress can be expected much earlier.

The presence of systemic disease as a source of discomfort can be only too plainly portrayed in the arteriosclerotic, the diabetic, the nephritic, the cardiac and the arthritic patient. As a point of emphasis on the importance of a careful consideration of the influence of systemic disease, suffice it to say that the most brief discussion of the manifestations of

constitutional disease in the foot would require a prohibitive length of time

The chief elements presented by the feet alone in the production of pain can be classed as muscular disturbances, neglected sprains, inflammatory lesions and deformities. The influence of wearing apparel, since it is a most constant covering of the foot and since its shape is governed by style instead of the anatomical characters of the foot, is one of the most common factors in the production of pain and deformity. The present-day feminine type of style shoe with its high, narrow, wobbly heel, short, low, pointed vamp, is probably responsible for more discomfort than any other single factor.

The cases presented in this clinic represent disturbances in the anterior part of the foot. The pain in such instances may vary from a mere feeling of discomfort, numbness, or dull ache, to a sharp stabbing or burning or even a severe cramplike nature. It may be ill defined and vague in its distribution over the region of the ball or it may be definitely localized in the region of one of the metatarsophalangeal joints or in one of the toes. Too, it may be associated with the symptoms of inflammation or deformity.

The treatment for the relief of pain in every instance depends upon the accurate determination of the cause of the pain and the elimination of that cause. In the obese individual one must reduce the weight, in the weak foot one must institute correct postural habits in standing and walking along with corrective exercises, while in the arthritic, the arteriosclerotic and the diabetic patient one must direct the treatment at the general as well as the local condition. A careful study of the causes of painful feet will convince any competent physician of the fallacy of focusing the attention upon the feet alone in an attempt to establish satisfactory remedial measures.

**Case I.**—Mrs H. S., aged thirty-six, weight 206 pounds, height 5 feet 3 inches, housewife. On April 2, 1935, the patient complained of extreme soreness on the ball of the left foot in the region of the base of the second toe. The disturbance was of two years' duration and was always considerably

worse after a great deal of walking. The annoyance of the pain had become so severe the patient volunteered the statement that she wanted relief regardless of the type of treatment or shoe required. Examination revealed tenderness on pressure beneath the second metatarsophalangeal joint, along with limitation of plantar flexion of all the toes. The shoe constantly worn by this patient had a very narrow heel  $1\frac{1}{2}$  inches high with a short low-pointed vamp. An outline of the sole of the shoe showed it to be much narrower than the bearing surface of the foot. Until one month before examination the patient had weighed 223 pounds. According to a standard weight chart her weight should have been 140 pounds. A diagnosis was made of foot strain manifested by pain in the anterior part of the foot.

The factors in the production of the pain were determined to be 83 pounds of excess weight until one month before examination and a high heel shoe with a too small compartment for the toes. A shoe heel  $1\frac{1}{2}$  inches high continually throws the weight forward on the anterior part of the foot and when the toes are crowded together by the front part of the shoe their normal activity is greatly limited and limitation of motion and muscular atrophy soon follow. The function of the toes in aiding the foot in walking is seriously impaired.

The management of this case consisted of weight reduction, a low broad heel on the shoe and a wide compartment for the toes, along with exercises. Relief is invariably the rule when such changes are made. Because of the acute symptoms, it is often necessary to keep the patient off the feet for a short time in order to allow the inflammatory reaction from injury to subside. Pain in the forepart of the foot in such cases as the above may be a mere feeling of discomfort, a dull aching pain, a burning sensation or even a sudden spasmodic cramplike pain in the region of the ball and in conjunction with it there may be a numbness or tingling sensation in one of the toes.

Case II—Mrs A. D., aged fifty-five, weight 110 pounds, height 5 feet 5 inches, housewife. On August 10, 1934 the patient complained of an aching pain across the ball of both feet. The pain had been present for two or three months but was much worse the last fourteen days and was most noticeable in the left foot. The removal of her shoes gave no relief. Upon retiring at night the pain slowly subsided. She also complained of stiffness in her knees which was most marked after rising from a sitting position. Examination of her mouth revealed 3 gold crowns. x-Ray examination of these teeth showed all to be devitalized and abscesses at the bases of 2 of them. Manipulation of the great toe of the left foot produced pain in the region of the metatarsophalangeal joint. Her shoes were the typically conventional type with short pointed vamps and narrow heels 2 inches long.

The treatment of this patient consisted of rest periods off the feet, contrast baths, removal of all known infected teeth and a low heel, broad toe shoe

On August 31 1934 she reported back for observation having carried out all previous instructions given her She was greatly improved

The impression of Case II was arthritis of the feet in the presence of foci of infection and faulty shoes

Trouble is almost inevitable when the foot has added to its normal burden the presence of arthritis and a shoe that interferes with its function as a weight bearing organ This patient at the age of fifty five years had arthritis in the feet and knees in the presence of definite foci of infection in the mouth The management of such a case for the relief of pain in the feet certainly necessitates the installation of anti arthritic measures which, of course, consist of rest, improvement of function in the feet along with physiotherapy and attention to all foci of infection There are certain factors one must seriously consider in attempting to obtain relief for the patient with arthritis in the feet. In obese individuals, the extra burden of excess weight upon the inflamed joints must be removed to the greatest extent possible It is need less to mention the practical good that comes from the removal of foci of infection throughout the body The common conventional shoe so universally worn by women with its high, narrow, unstable heel and narrow pointed vamp must be promptly replaced by one that will permit more normal function in the foot and more normal distribution of the weight The too thin sole that allows trauma from the hard present-day pavements is better replaced by a thicker one that affords more protection to the inflamed joints of the foot In mild cases of arthritis where a flimsy type of shoe is worn, a good strong shoe with a stiff shank will often give enough relief as a single factor to increase the patient's comfort many degrees Those individuals who have occupations that keep them for many hours upon their feet need in addition to other antiarthritic measures, rest periods during the working hours In many cases it may be necessary for the patient to arrange

to be absent from work for several days at a time to give absolute rest to the feet. People that require this change, especially in the present economic situation, are oftentimes the most difficult to manage, because the very thing they need most, rest, is the hardest thing for them to obtain. In stubborn cases a change of occupation is unavoidable.

**Case III**—Miss B. T., aged twenty-four, nurse. On June 30, 1933 this patient complained of extremely painful corns on the toes of the right foot. Examination revealed the following facts: A hard corn on the lateral surface of the fifth toe, a soft corn on the median surface of the fifth toe and a soft corn on the lateral surface of the fourth toe. She was having excruciating pain from these common lesions and was at a total loss about what to do, since she had tried this and that conventional shoe and had been here and there to have her corns trimmed and padded and treated. Her toes were crowded into the front part of an ill-shaped common style shoe with 1½-inch heel. Well-defined ridges were present in the skin of the toes along their dorsal median and lateral borders due to compression of the toes in the shoe.

This patient was immediately relieved when her shoe was cut to relieve the pressure. Hot moist dressings were used to allay the acute inflammatory reaction and when the inflammation subsided she was fitted with low broad heel shoes with wide toe compartments permitting no pressure on the toes. Subsequent contact with this patient revealed the fact that she was in perfect comfort as long as she wore the prescribed shoes.

Temporizing with corns adds nothing permanent to the patient's comfort and casts its reflection upon the physician consulted. The front part of the average foot is wide while the front part of the average woman's shoe is narrow and pointed, thus directing the toes toward each other instead of allowing them to maintain their normal parallel positions. Case III represents one of the most common and prevalent kinds of foot discomfort and one of the most improperly and inefficiently treated conditions with which the physician has to cope. The treatment is simple and the physician who has the interest of his patient at heart should see to it that he personally supervises the fitting of shoes on such a patient. While the treatment may be distasteful to the patient's pride, it behooves the physician to be very firm in his assertion of the necessity for a shoe that is not only wide enough, but of

the right shape, so that it does not throw the weight forward on the foot, thus crowding the toe down into the dart-shaped anterior part of the shoe. One must forever keep in mind that pressure is the outstanding cause of corns and until it is effectively removed improvement cannot take place

**Case IV.**—Mrs P M aged twenty five, weight 160 pounds, height 5 feet 7 inches. On October 1 1934 the patient complained of pain across the ball of both feet and pain in the region of the median part of the longitudinal arch. On June 2 1934 she had been confined. She weighed 125 pounds before becoming pregnant. During pregnancy her weight went from 125 to 185 pounds. Since delivery she has weighed 160 pounds. Her foot discomfort began during the puerperium after she was again on her feet. She was in bed eight weeks after her delivery due to venous stasis in the left leg. Her present discomfort is most severe in her left foot. When she came in for treatment she wore a conventional type shoe with a heel  $1\frac{1}{2}$  inches high with a round toe.

Here we have an individual who gained 60 pounds during pregnancy and then following labor was confined to bed for eight weeks. The gain in weight represents an increase in the load upon the feet. Confinement in bed because of illness or childbirth allows atrophy of disuse to take place in the muscles. When the patient again takes to the feet with the increased weight and the weakened condition of the leg muscles foot strain manifested by pain in the region of the instep or up the outer side of the leg is a common sequence. The muscles of the legs that control the feet must be prepared for the increase in weight by proper shoes and exercise during pregnancy. Following delivery it is very important that the burden put upon the feet be gradual and that the patient be instructed in correct postural habits and exercises. Support for a temporary period in the shoe is often of advantage. This may be accomplished by the fixing in the shoe of pieces of felt that fit under the region of the longitudinal arch.

Similar discomfort to that described above is often present in children when they again take to their feet following the acute diseases of childhood. However, the child often limps and favors the part for a while before actually complaining of pain. Because of the commonness of foot strain in children due to faulty posture in standing and walking, it is imperative that the attending physician give instructions to the parents about the vigilance over the child's feet when it gets up after an illness.

**Case V.**—Mrs. I L aged fifty-eight housewife. On February 21, 1934 she complained of painful enlarged great toe joints which had been present for fifteen years. She also gave a history of arthritic symptoms in the

hip joints Examination revealed marked limitation of motion in both great toe joints in all directions but especially in dorsal flexion After careful consideration of her general condition a diagnosis of hallux rigidus due to productive arthritis of the first metatarsophalangeal joints was made Anti arthritic measures were instituted and for local treatment the patient was fitted with a pair of low, broad heel shoes with broad toe compartments Underneath the great toe joint and just distal to it a wedge of leather  $\frac{1}{2}$  inch thick was placed in the sole of the shoes After a preliminary rest period to quiet the acute symptoms of arthritis the patient was allowed to walk about in the newly prepared shoes Since she has worn the modified shoes, she has reported back a number of times in the last thirteen months and in each instance she reported that she had never been so comfortable for years

The wedge in the sole of the shoe stiffens the sole and limits motion in the great toe joint In such cases of hallux rigidus, Whitman mentions the use of the wedge on the inner side of the sole to stiffen it and limit motion in the great toe joint

**Case VI**—Mr L B, aged fifty-five On January 28, 1933 he appeared for treatment of a "bunion" on the right foot which he said had been present for several years but had become inflamed and painful the last six weeks Examination revealed enlargement of the great toe joint of the right foot with a reddish circular area on the skin of the median part of the joint The first impression of this case was that the lesion presented was due to a tight shoe Warm moist applications and rest without the shoe made little change in the condition A thorough examination of the patient was then made and revealed the following findings Blood pressure, systolic 162, diastolic 88, blood sugar 333.2 mg per 100 cc of blood, urine showed 1 plus sugar, condition of cardiovascular system showed evidence of arteriosclerosis Prompt attention was then given to his diabetes and arteriosclerosis with the result that he improved He was not seen for two years He returned for further treatment on April 23, 1935 At this time there was definite gangrene of the toes of the right foot On May 3, 1935 his right leg was amputated because of spreading gangrene

The significance of this case is the fact that the patient totally unaware of the presence of arteriosclerosis and diabetes was seeking relief for a painful disturbance in the foot It shows emphatically the absolute necessity for a complete physical checkup in all cases presenting painful foot disturbances

**Case VII**—H B, male, aged thirteen, weight 143 pounds, schoolboy On April 29, 1933 this boy was brought in by his father because of a painful swelling on the dorsum of the left foot over the second and third metatarsal

bones. The condition had appeared gradually and was present for about two weeks. There was no history of injury. Because of a disturbance in the left hip joint this patient had placed no weight upon the left foot for many months prior to December 3, 1932. From December 3, 1932 to April 29, 1933 he had been walking. Examination revealed an overweight boy thirteen years old with a tender swelling over the dorsum of the left foot. x Ray examination revealed a fracture with callus formation just distal to the middle of the second metatarsal bone. The diagnosis was march foot resulting from overuse of the foot following a long period of inactivity with the contributory factor of excess weight. The treatment consisted of rest off the foot and later exercise and gradual increase in the use of the foot. Complete recovery resulted.

A number of cases of march foot were reported by Speed and Blake in 1933, but in each instance the patient was over twenty years of age. They explained that "The condition has been well recognized for many years, especially by the German and French military surgeons, who frequently encountered it among their troops after strenuous duties or long marches."

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## CLINIC OF DR G K FENN

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#### PAIN SIMULATING THAT PRODUCED BY CORONARY DISEASE

IN collecting material to show causes for pain apart from coronary disease, one is astounded by the multiplicity of causes that present themselves. In many instances the pain is the result of a disturbance of the coronary circulation, but without coronary disease as such disease is usually considered. The latter causes have no place in this discussion, and I shall present no case records bearing upon them. I feel constrained to mention a few of them, because of their frequent occurrence. Hypotension is often accompanied by an anginal type of distress. As the coronary circulation is largely dependent upon the blood pressure, such distress is not surprising. Hypothyroidism is frequently attended by similar distress, even when the blood pressure is relatively undisturbed. The reason here is not quite so clear, but probably arises from the lowered metabolism. Neurocirculatory asthenia or the effort syndrome may be associated with severe pain, sometimes simulating coronary occlusion. Severe anemia may produce anginal pain. Some years ago anginal pain was a common symptom in pernicious anemia. This distress is doubtless due to anemia of the heart muscle as a part of the general anemia.

There are other causes of pain that have their origin in the heart or coronary circulation, but with these few examples we shall proceed to a consideration of causes that are entirely extracardiac. One of the most common sources of confusion in this regard is the pain arising from osteo-arthritis of the spine. Scarcely a week goes by that one is not confronted with the

necessity of differentiating between this disorder and angina pectoris. This arthritic pain may be referred over a large area of the chest, and to the shoulder, and greatly resemble anginal pain. It comes on as a result of exercise, occurs usually in patients past middle life, both of which add to the similarity. Suspicion will be aroused, however, by the fact that the pain is more constant than anginal pain. The pain is accentuated by exercise involving the upper extremity and is accentuated by standing or lying in certain positions, while it is relieved by assuming other and often more uncomfortable positions. These observations will point to the true source. Examination will show a relatively normal cardiovascular system, and usually evidence of arthritis elsewhere. An x-ray of the spine will show pathology in that region.

**Case I.**—A woman of sixty-eight years gives a history of pain in the left chest and shoulder. This has been present intermittently for six years. She notes the pain is worse for several days after exercise such as housecleaning. There are times when the pain is absent for weeks, but when it reappears it is constantly present in varying intensity for days at a time. Sleeping on the left side seems to aggravate the pain, but sleeping or lying in bed on the back with the left arm extended above the head gives relief. This patient has been told she has angina pectoris, and has been receiving treatment. Examination shows cardiovascular system that is relatively normal for the age. There is a bit more than ordinary of arteriosclerosis. Exercise, such as walking up a stairway, brings no immediate increase in pain. There is evidence of considerable bony enlargement in the fingers and toes. An x-ray of the spine shows spicules and bridging of the dorsal vertebrae. Salicylates temporarily relieve the pain. Three years later the pain is unchanged or slightly better, and the patient has recently successfully combated a bronchopneumonia, a fair accomplishment for a patient of seventy-one years and a fair argument for a pretty sound cardiovascular system.

*Acute fibrinous pericarditis* with its attendant pain, shock, fever and leukocytosis and elevation of the pulse rate, may easily be mistaken for coronary disease.

**Case II.**—A young woman of twenty-six had been subjected to an abdominal cesarean section because of an acute toxemia of pregnancy. There was an immediate relief of the toxemia and seventeen days following the operation the patient was enjoying excellent health. On this day she was seized with a sudden severe pain in the precordial region. The pulse rose rapidly to 130, the patient became pale and slightly cyanotic. The temperature rose to

102 F., and the following day there was a marked leukocytosis. No friction rub was heard. The picture closely fitted that of a coronary occlusion except for one symptom. There was rather severe pain along the left trapezius ridge. This did not rule out coronary pathology but it raised a strong suspicion of pericardial or pleuropericardial inflammation. On the following day there was a considerable increase in cardiac dulness with signs of pericardial effusion. *x* Ray confirmed the heart size and a diagnosis of pericarditis was justified. The patient made an uneventful recovery.

*Pulmonary embolism* when not immediately fatal produces a train of symptoms strikingly similar to those of coronary occlusion.

*Case III.*—A woman of thirty-two who had rheumatic carditis with mitral stenosis since childhood came in because of tachycardia and weight loss. In addition there was great fatigue and occasionally a slight elevation of temperature. She was married and had gone through two pregnancies without incident. A subacute bacterial endocarditis was suspected but the suspicion could not be confirmed. The patient continued to grow worse and a short time later it was discovered that she was again pregnant. A therapeutic abortion was decided upon and she was hospitalized for this procedure. At three o'clock in the morning of the day set for operation she had a sudden excruciating pain in the left chest. This was accompanied by an extremely rapid pulse 160 to 180, marked dyspnea, pallor and cyanosis. She was extremely apprehensive and was quite convinced that she was dying, a symptom so common in coronary disorders. A diagnosis of coronary embolism was made, assuming that there was present the endocarditis which had been suspected. A pulmonary embolism was considered but we lacked a good source for the embolus unless we assumed a right heart endocarditis. A few hours following the onset the patient spat up blood. This would fit into either picture. The pain persisted for several hours, and on the following morning a definite area of consolidation was made out in the left upper lobe posteriorly. This was most confusing until about thirty hours after the onset she began to bleed from the uterus. We were now furnished with the source of the pulmonary embolus which the condition proved to be. She aborted spontaneously and had as a further complication a thrombophlebitis of the left leg but made a good recovery.

There is a certain variety of pain associated with mediastinitis that occurs uncommonly but is very striking when encountered. I have seen but three cases of such pain. This phenomenon occurs in connection with mediastinal adhesions about the base of the heart. In my own cases it has been associated with adherent pericardium of rheumatic origin. The *x* ray shows a partial obliteration of the upper part of

the mediastinal cavity. My cases have all occurred in individuals under twenty-five years of age. The pain is sudden in onset, very severe, and is located beneath the sternum. It has a typical anginal radiation and is associated with dyspnea and pallor. I have not seen cyanosis. The attacks of pain are paroxysmal in character and are rather evanescent and seldom last more than ten to twenty minutes. The patient is rather prostrated following the attack, but is relatively free from pain. They seem most likely to occur at night. In two of these three patients the attacks became progressively more infrequent and finally disappeared. I do not know the outcome of the third case. I have been told that similar attacks of pain occur in certain instances of high-grade mitral stenosis with great dilatation of the left auricle, but I have not encountered such patients.

*Spontaneous pneumothorax* with sudden onset will occasionally offer a diagnostic problem. This accident is commonly attended by severe pain along the costal border on the side in which the rupture occurs. In left-side pneumothorax it may resemble the pain of coronary occlusion. The acute pain is frequently succeeded by a sense of oppression that adds to the confusion. The onset of the pain is followed by a sudden acceleration of the pulse, a fall in the blood pressure, pallor, cyanosis and symptoms of shock. Dyspnea may be most severe. Examination of the chest will ordinarily reveal the cause of the difficulty. Pneumothorax with insidious onset may show no distinctive findings for several days. With sudden onset the classical hyperresonant or tympanic chest may not be found. Air in the pleural cavity under certain degrees of pressure will produce a high-pitched tympanic note that resembles dullness. The ruptured side, however, will be found fairly well fixed. The breath sounds will be impaired, and the heart dullness will be obscured or pushed to the opposite side. The x-ray will substantiate the diagnosis.

It would seem difficult to confuse traumatic pain with that of cardiac origin and yet we have in our records a case of a physician who came in with his own diagnosis of angina

pectoris or coronary occlusion. He had a steady pain in the left chest. This pain had persisted for almost two days. It was present when he awoke one morning and became worse on exercise. This increase in pain on exercise was a constant feature. There was no shock or disturbance of cardiac rate or rhythm, but he became nauseated as the pain increased. Examination showed a sound cardiovascular system, but a great deal of tenderness over a limited area in the left chest. The  $x$  ray revealed a fractured rib. It was impossible to determine how or when the accident occurred.

*Fibrinous pleurisy* not infrequently has as its initial symptom sudden, severe pain. This pain may originate in the chest or it may be first felt in shoulder or neck, depending upon the site of the inflammatory process. There appears to be dyspnea, but in reality the breathing is shallow and rapid rather than difficult. While the steady boring pain may resemble that of a coronary attack, the lancinating pain occasioned by a deep breath, will direct attention toward a fibrinous pleurisy and a friction rub will usually be heard if sought for at frequent intervals.

In a similar manner does lobar pneumonia occasionally introduce itself, with a steady boring chest pain of sudden onset. This together with the rusty sputum and the increased respiratory rate often raises a suspicion of coronary disease. The acute infectious nature of the disorder is usually recognized within the first twenty four hours, and in the majority of cases unmistakable physical signs of pneumonia will appear by the end of the first day. This latter statement is by no means always true. We have numerous records in which the entire pneumonia showed a minimum of physical signs, and the  $x$  ray findings were responsible for the diagnosis.

It would seem almost unnecessary to mention the pain of luetic aortitis and aneurysm in this connection, yet these conditions have been confused with angina pectoris and coronary disease. A careful physical examination will serve to differentiate.

So far I have presented records of pathology in and about

the chest as a cause of anginal pain. I have a few more illustrations of pathology outside the chest that has produced this picture. In a small percentage of peptic ulcers the first indication of their presence will be a perforation into the peritoneal cavity. This accident may be mistaken for a number of things, among which is coronary occlusion. The sudden onset of excruciating pain high in the epigastrium and radiating upward, the profound shock and the elevation of the pulse rate often justify this suspicion. Subsequent developments when sought for will correct the error.

**Case IV**—A man of forty-two years, who had an old rheumatic carditis, was suddenly seized with excruciating pain high in the epigastrium. The pain radiated upward into the chest and into the right shoulder. When first seen he was in a state of shock, was pale and prostrated, and the pulse was 120 and thready. Blood pressure had fallen from 130 to 90 mm. Pain was severe and continuous. Respirations were shallow and rapid. There was a suspicious rigidity of the abdominal muscles but not enough to prevent a diagnosis of probable coronary occlusion. Four hours following the onset of the pain the abdominal rigidity had become unmistakable, and in six hours it was sufficient to change the diagnosis. By this time the pain had begun to move downward into the abdomen. The temperature did not rise above 100.5° F, and the highest preoperative white count was 11,000. Seven hours after the initial attack the abdomen was opened and a perforated ulcer was found. The patient made an uneventful recovery. This patient had been under observation for several months preceding the perforation and the presence of ulcer was not suspected.

Another patient that caused a few uneasy days was a man who was hospitalized because of an accident. There was no previous history of heart disease, but while lying in bed he developed an annoying pain in the left chest and shoulder. Realizing the frequent occurrence of thrombosis in patients who are bedridden, the question of a coronary thrombosis was immediately raised. Probably because of the frequent examinations of the cardiac area, the patient became "heart conscious," a condition which had not existed before. Nothing was found to substantiate the diagnosis of coronary thrombosis, and the condition subsequently proved to be a subdeltoid bursitis.

Latterly, we are likely to look upon the diagnosis of "acute

indigestion" with some suspicion. It is true that many, perhaps most, deaths from "acute indigestion" are coronary accidents, but we must not overlook the fact that minor disturbances of the gastro-intestinal tract may produce symptoms closely resembling coronary disease. The most severe abdominal pain may result from the distention of the spastic colon. Gas imprisoned in the splenic flexure will produce pain radiating upward into the chest and in addition will produce dyspnea, pallor and elevation of the pulse. To add to the difficulty of differentiation it must be remembered that in the anginous patient, gaseous distention is capable of producing the attack of angina pectoris. In such situations one is confronted with a delicate diagnostic problem.

Finally, diaphragmatic hernia has been a source of anginal pain. In those cases with which I am familiar the hernia has been situated at the esophageal ring. The x-ray has confirmed the diagnosis. In the cases of Drs. Portis and Bettman surgical repair of the hernia has abolished the anginal symptoms.

This list, I believe, represents the most important situations in which coronary disease may be justly suspected. I would call your attention to the fact that there are pitfalls for you on both sides of the diagnostic fence. Treating a coronary occlusion as an acute fibrinous pericarditis and allowing full liberty with the subsiding symptoms would be likely to result badly for the patient. On the other hand, treating a perforated ulcer as a coronary occlusion would almost certainly result fatally. A correct differential diagnosis is a most comforting thing.





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#### TREATMENT OF ANGINA PECTORIS

BEFORE discussing the treatment of angina pectoris it will simplify the problem if we first make clear just the clinical syndrome we are referring to when we speak of angina pectoris, and if we review briefly some of the conditions predisposing to the attacks of anginal pain which have a direct bearing upon the treatment. We must also make certain that angina pectoris is clearly differentiated from coronary thrombosis. Just as in coronary thrombosis, the outstanding symptom is pain and pain which differs in no way from the pain of coronary thrombosis, except as to its duration. As in coronary thrombosis, also the pain is due to a blood supply to the heart muscle which is inadequate for its needs.

In coronary thrombosis there is an actual occlusion of one of the coronary arteries by a thrombus within its lumen. There results an interference with the blood supply to some portion of the heart muscle which persists. The extent of the area involved and the amount of anatomical damage and its permanence depend upon the position of the thrombus in the arterial tree and upon the ability of anastomoses, collateral circulation and the thebesian circulation to provide some degree of blood supply in the muscle affected. The amount of damage done may be only slight in some few favored cases, but it is usually enough to produce continued subjective symptoms and to impair the function of the heart to the extent that there are objective evidences of cardiac insufficiency. In some cases the amount of damage done is such as to be im-

mediately incompatible with life or to produce a fatal result in a very short time

In angina pectoris the attack of pain is not due to the anatomical occlusion of any particular vessel immediately preceding the attack. The adequacy of the coronary circulation in the hearts involved varies through a wide range, but it is not permanently altered to any appreciable degree by any one attack. Whatever the condition of the coronary circulation may be, the pain occurs when the metabolic needs of the heart muscle are increased out of proportion to the blood supply available at the moment. The attack is of short duration and ceases when the demands upon the heart muscle are decreased, or when the blood supply is adjusted to meet the demands. Between the short attacks the patient is in what is a normal condition for the individual.

There are not any characteristic objective findings. It is probable that repeated short attacks may in time result in permanent myocardial damage. It is also probable that an attack may persist long enough to result in ventricular fibrillation and death.

There is a zone of common ground of indefinite breadth where it is difficult to differentiate between the two conditions in the first attack or in some individual later attacks. A coronary thrombosis may occur with very slight or moderate pain, or pain of very short duration with very little in the way of objective findings and still have even fatal consequences. The final outcome of recurring attacks of angina pectoris is very likely to be a coronary thrombosis and such a coronary thrombosis must be kept in mind in observing the recurrent attacks. In any case of doubt, the prudent procedure is to treat the case as one of coronary thrombosis until it has been proved otherwise.

Aneurysm, or a syphilitic aortitis without aneurysm, may be productive of substernal pain and should be differentiated. As a rule, the pain is quite different and whether only a dull ache or a very severe pain, is more prolonged and is not a paroxysmal attack precipitated by some definite cause. An-

ginal attacks may co-exist, especially if an aortic insufficiency is present. Other mediastinal conditions, as tumor or mediastinitis, may cause substernal pain. When such known pathological changes are present, the diagnosis should be that of the condition present, and treatment should be that which considers that condition.

Attacks of angina pectoris usually occur under conditions which entail additional work for the heart muscle. Exertion is the outstanding exciting cause. The amount of exertion is not constant for the individual and will vary in the presence of various physiological and psychological conditions. Attacks are most frequent after meals because of the increase in cardiac output at that time or this increase plus that produced by exertion or emotional excitement. Heavy or indigestible meals predispose to attacks.

The attack may follow any meal or it may be especially after breakfast in the morning when the blood pressure is lower, or after dinner at night when the heavier meal or the fatigue after a busy day may be an additional factor.

The attacks in some cases may come on only during the night and awaken the patient from a quiet sleep. Such attacks usually occur in the early morning hours when the blood pressure reaches its lowest level for the twenty-four hours.

In some patients the attacks may come on without apparent cause when the patient is resting quietly, but usually some relation to some exciting factor may be found.

A large nervous element in the precipitation of attacks has attracted attention since the time of Heberden, who classified angina pectoris with "the incubus, convulsive asthma, numbness, hypochondriac languors, and other ills justly attributed to the disturbed function of the nerves." In support of this he cited the "sudden manner of its coming on and going off, the long intervals of perfect ease," the "influence of passionate affections of the mind," and the number of years it will continue without otherwise "disordering the health."

The present-day observer cannot fail to be impressed, as Heberden was, with the importance of these causes of ner-

vous origin His nomenclature may be different and he now recognizes that the factor of nervous or emotional origin produces its effects by means of physiological processes which increase the work which the heart must do in proportion to its blood supply or decrease the blood supply while the work which the heart is doing remains constant Nevertheless, the importance of these causes remains

Any of the emotions or mental excitement of any nature may precipitate an attack Anger is a frequent cause A patient may have frequent attacks on a day when the worry and strain of business life is increased, and few or none on a day when all is going well Angina pectoris has frequently appeared in those previously free from attacks because of anxiety or of grief or worry over business reverses Attacks may appear under emotional strain and disappear when the strain is removed The same man who has daily attacks when he has the responsibility of an executive position to worry over may go on a vacation which calls for much more physical exertion than does his daily life at work and not have a single attack Or the man who cannot walk from the station to his office without having to stop because of anginal pain, can play golf in comfort

In these cases which show recurring attacks of anginal pain, the heart shows varying degrees of pathological change from a very great deal to those which are very slight or those which would be expected at the age Between these two extremes is every possible gradation In a portion of the cases the symptoms can very definitely be ascribed to pathological changes and in another part of the cases it is very difficult to ascribe the symptoms to pathological changes alone and some physiological factors must be considered In a very small proportion of cases qualitative changes in the blood are a cause of anginal pain

In part of the cases there are arteriosclerotic changes in the coronary arteries sufficient to interfere with the blood supply to the heart muscle The blood supply may be sufficient for ordinary needs in the presence of these changes, or

for the needs of the heart muscle when the body is at rest. But when the heart muscle is called upon to do additional work, as during exertion or during nervous or emotional stress, or during the increased cardiac output occurring during digestion, the arteriosclerotic blood vessels are unable to meet the increased demands of the heart muscle for blood, and pain results. It is in such cases, also, with advanced vascular degeneration that attacks may come on during the night, in the early morning hours, during sleep, when the blood pressure is lowest.

Since the coronary flow is proportionate to the blood pressure, the higher blood pressure present during the day is able to maintain an adequate flow in spite of the vascular changes which are present. When the blood pressure falls during the night, the coronary flow, decreasing with the decreased blood pressure, becomes inadequate to compensate for the vascular changes. In those cases where attacks come on mostly in the morning, the lower blood pressure of the early morning is also a factor. The coronary flow is adequate when the patient is at rest, but not for the additional demands of exertion or those occurring during the digestion of breakfast.

In cases where there are vascular pathologic changes present in the coronaries not sufficient to cause symptoms under ordinary circumstances, a toxic thyroid, by its increased demands upon the work of the heart, may be the determining factor in producing attacks of angina pectoris. The increased emotional instability produced by this condition affords an additional factor.

Aortic insufficiency also furnishes an anatomical basis for anginal pain in some cases. The coronary flow varies with the blood pressure, but especially with the diastolic pressure. In aortic insufficiency, the low diastolic pressure may be such as to decrease the coronary flow to a point where it is insufficient for the work being done by the heart or insufficient in the presence of sclerosed arteries. The low diastolic flow is compensated for in part in most cases by an increased systolic pressure and to some extent by an increased heart

rate These points must be kept in mind when treatment is planned

Aortic stenosis is frequently accompanied by attacks of anginal pain There is usually some degree of insufficiency present which in this case cannot be compensated for by a high systolic pressure or an increased pulse rate In addition there is the increased work demanded of the hypertrophied left ventricle by the stenosis

Old healed infarcts from a coronary thrombosis in which an adequate circulation has not been restored by anastomosis or collateral circulation may be the basis for recurring attacks or angina in some cases In part of these cases we have a history of an initial attack of coronary thrombosis and in others a healed infarct may be found at autopsy which cannot be placed in the history

Another anatomical basis for a decreased coronary flow is the partial occlusion of the mouth of one of the coronary arteries by changes in the aorta, especially changes due to syphilis In a few cases there are congenital anomalies of the coronary arteries which may afford an adequate flow in early life, but a flow which becomes inadequate in the presence of the vascular changes which occur later in life

It would appear certain that anatomical changes in the heart or its vessels constitute the chief basis of anginal attacks and play a part of varying importance in most of the cases But such anatomical changes cannot be the sole factor We still have to explain the attacks occurring in cases whose hearts are normal for their age It is difficult also to explain the influence of the nervous and emotional background upon increased circulatory demands alone Anatomical changes in the arteries do not explain why a man who has several attacks a day while at work has none upon a vacation where he is more active, but free from responsibility and worry, and why the attacks recur when he again returns to work Certainly the vascular pathology does not change back and forth There must be some other factor present We cannot take the time to discuss this fully here, but it would seem to be connected

with an over-labile autonomic system. It is possible that it is a question of a generalized instability of the vasomotor system and an over-response to normal stimuli which throws an increased load upon the heart. It is possible also that a reflex vasoconstriction of the coronaries is responsible for the attacks in some cases or an increased tonus of the coronaries with inhibition or failure of the vasodilator response. Such a mechanism would not be normal and would not be to the biological advantage of the organism. The organism, however, is not living under conditions of life which are of biological advantage or it may not be a normal organism. The bronchoconstriction of asthma is not advantageous to the organism, but it does occur. In our animal ancestors one might feel that an autonomic system with a lowered threshold to stimuli and an over response might have been of advantage under conditions of fatigue, but not with a response which involved a vasoconstriction of the coronaries. Such conditions, however, must involve the autonomic system as a whole and cannot be selective of any one function.

Whatever the mechanism, whether active changes in coronary caliber or some response which throws an increased load upon the heart, its association with an overlabile and over-active autonomic system would explain a great deal. It would help to explain the more frequent occurrence in the tense, nervous type of individual who has what Dr. Stuart Roberts very aptly terms the "spasmogenic aptitude." It would explain the more frequent occurrence of angina in business and professional men working under strain, and the effect of overwork, worry and fatigue with inadequate leisure, and in adequate or tiring vacations, for the autonomic system becomes more unstable with fatigue and with chronic fatigue or "staleness." It would explain the occurrence of anginal pain in the so-called "effort syndrome" for whatever that symptom complex is or is not, there is present a lowered threshold to autonomic stimuli and an over-response to those stimuli. It would explain the increased incidence of angina pectoris with



our changed social conditions, with lives of increased strain, and less real rest and relaxation

Just how much of a factor tobacco is in producing attacks is uncertain. On the whole it probably has little influence. There are some cases which are associated with what is for the individual an overuse of tobacco. Smoking does have a vasoconstrictor effect upon the peripheral vessels and in experimental animals even very small doses of nicotine have a vasoconstrictor action upon the coronaries.

Digitalis administration is responsible for anginal pain under certain conditions in patients with a normal rhythm, and more rarely where more than the optimum dosage is given in auricular fibrillation. A decreased coronary flow has been observed when digitalis is administered to experimental animals. There are other variables determining the coronary flow volume, and while a decrease is observed more frequently than not, it does not always occur. It is assumed to be due to a coronary vasoconstrictor action of the digitalis. Increase in tonus of the heart muscle is probably not responsible for the decreased flow as the flow remains constant under changes in tonus produced in other ways.

In addition to a blood supply to the heart, which is quantitatively inadequate for the needs of the heart muscle, we find some cases where the attacks are consequent upon a blood supply that is qualitatively inadequate, or qualitatively inadequate in the presence of some degree of arteriosclerosis in the coronary vessels.

One such qualitative factor is the decreased oxygen carrying power of the blood in pernicious anemia. In the cases reported by Herrick, who first called our attention to anginal pain in pernicious anemia, arteriosclerotic changes were also present in the coronary arteries. The coronary changes are such as to allow an adequate supply of oxygen with blood of normal oxygen carrying power, but do not admit of sufficient oxygen in the presence of the impaired coronary circulation.

It is not only a question of sufficient oxygen supply. The heart muscle needs other materials carried by the blood, and

sugar is of especial importance Cardiac pain due to hypoglycemia following the use of insulin has been discussed by several writers in recent years Strouse and his associates have called our attention to anginal pain with a low blood sugar, following an inadequate carbohydrate diet Sippe recently reported 4 such cases with a low blood sugar on their normal diet Relief followed upon glucose given by mouth Sippe also reported a case with a normal blood sugar, but a ketosis Improvement followed upon an antiketogenic diet

Anginal pain may also occur in cases with an abnormally high blood sugar but in whom a deficient insulin supply does not admit of its proper utilization Such cases are relieved by the proper dosage of insulin

While I have spent some time in going over the conditions which may constitute the basis for the recurring attacks of anginal pain, I have really gone over the ground much more hastily than I could wish It is obvious that treatment is not the simple matter of prescribing some one remedy Just what we are to do to help our patient will depend most of all upon a proper evaluation of the points which we have just considered as well as many other points which we have been forced to omit for lack of time.

The first requisite is that the physician himself should recognize that there is always something which he can do to help and that very often he can do a great deal He cannot do it by writing out a routine prescription The physician must obtain personally a careful history and be guided by what he learns from the history in each individual case There can be no routine procedure. He must give time and thought and understanding and patience No amount of careful "work up" can replace the thoughtful understanding of one man who is in a position of leisurely and friendly contact with the patient and who is ready to assume responsibility for the future welfare of the patient

A great deal of the patient's future depends upon what the physician says A few years ago Rudyard Kipling in

addressing a medical audience spoke of the therapeutic value of words. And they have a great therapeutic value. In angina pectoris it is especially important just what you say to the patient and how you say it. There can be no fixed rule for this. Second only to the physician's knowledge of the science of medicine—in some cases even more important—is his ability to understand others, to appreciate what is going on in the patient's mind, to feel as he feels and to think as he thinks and to foresee the reaction to the spoken word. The physician must be able to "*se fourrer dans la peau d'autrui*"—to put himself into the other's skin. No matter what his anatomical background, the nervous element is always present in some degree and has as great an influence upon the patient's future welfare as it has had upon the genesis of his complaint. He must be given faith and confidence and encouragement, and at the same time be told enough so that he can best avoid attacks and prolong life. This is not an easy task.

It is rarely advisable to deny flatly to a patient that the pain of which he complains is anginal. There are some patients with whom such a flat denial is the wiser course and who are quite willing to assume that the pain is that of indigestion or of gas. If they will continue to think so, it is quite as well that they should, provided that the régime ordered by the physician is that which the actual condition requires and that they are kept under observation. But a great many of those who seek advice have had the doubtful advantage of our present-day popular education in medical matters. Daily perusals of health columns and other sources of information have already given them some idea as to what the basis may be. It is better to say what the pain is and to explain the condition in a way which reassures them than to lose their confidence by denial. Their confidence you must have and to have it and retain it, you must earn it. It is quite possible to discuss the condition frankly and at the same time to reassure the patient and to help him.

A bad prognosis should never be given, certainly not for

the patient's sake and also because you yourself do not always know sufficient in regard to the prognosis. Many a patient given a hopeless prognosis has been made infinitely worse by it and also many a patient given a hopeless prognosis is alive and well years afterward.

There will be a group of patients in whom there are symptoms other than the anginal pain, such as shortness of breath, dyspnea on exertion, and other subjective and objective evidences of impaired cardiac function. Part of this group will consist of those who have previously had a coronary thrombosis or in whom there is myocardial damage due to advanced coronary disease or other factors. Some of them may show very little in the way of objective findings and others will show definite findings of heart disease with or without congestive failure. Included in the group will be cases which show the resultant changes of rheumatic fever, of syphilis, or of renal disease. Here also will belong part of the cases of aortic insufficiency of either rheumatic or syphilitic origin. In all such cases, treatment must consider the pathological conditions present, their etiology, and just how they are interfering with normal physiological processes and functions. In a large part of this group, rest will be the first therapeutic indication, or at least some limitation of activity. It must be determined whether the infective process or other agent, as toxic thyroid, which caused the damage, is still active. If infection is still present or a toxic thyroid is active, that must be considered first of all.

In cases of syphilitic origin, it is best to proceed with considerable caution as regards treatment of the syphilis, and it is sometimes more prudent not to treat the syphilis at all. It is wise to bear in mind Dr. Hay's dictum and "treat the heart first, and not the syphilis." We will discuss this further when we are showing the cases.

There will be a large group of patients in whom the only subjective symptoms are the anginal attacks and in whom there are no characteristic objective findings. A part of this group may be found in perfect health aside from the anginal

attacks In another there may be some slight evidence of impairment of cardiac function In a portion of these patients some degree of rest may be advisable at the start In some this may consist of complete bed rest with a gradual resumption of activity This will have two objectives, to hasten recovery from fatigue and to permit possible recovery processes in the heart In some a continued limitation of activity will be advisable Just what to do in regard to rest is a matter of judgment in each case There will be some cases where the apprehension caused by such measures will more than offset any possible good results

Unless there are definite indications to the contrary, most patients are better if allowed to go about their duties and recreations about as usual with only moderate limitations It is better from a psychological standpoint and there is the possibility also that with a moderate load put upon the heart, further anastomoses and collateral circulation may develop In many cases the possible fall in blood pressure with prolonged rest is not to be desired There are obviously patients who cannot be allowed to continue their duties, such as those whose duties would tend to precipitate attacks and those whose sudden incapacity would endanger themselves or others

There is a great deal in the way of general directions for the patient which should be talked over with him in a quiet, friendly, unhurried and undisturbed visit The possible exciting causes for attacks, and especially those applicable in his case, should be explained to him As far as possible he should be instructed to avoid attacks, and to keep within the limits of the exertion which will bring on an attack If an attack does come on, he should be instructed to stop and rest until it wears off Unless the pain stops at once with the cessation of effort, he should take a *nitroglycerin* tablet There is always the possibility that some recurring attack may be a coronary thrombosis Because of this it is better that he should be advised that in the event of an attack which is more severe or which bears with it unusual symptoms, he should

return to his home at once in a manner entailing as little effort as possible, and advise his physician

He should be instructed in regard to the necessity of leading a quiet, calm and orderly existence, avoiding strain and hurry and anxiety. He will find that he can cultivate a spirit of philosophic calm much more easily than he supposes. Shorter hours of work and more rest are always advisable. Recreation should be such as to afford rest and relaxation and such as does not add to the nervous strain. When possible a quiet, restful vacation is always a good start on the treatment and is a therapeutic measure which should be frequently repeated. Frequent quiet week-ends in some small town hotel where he is not known and where he is out of reach of his business associates afford a very good rest. A short train ride to the hotel provides a sense of detachment from the worries of his daily life. He can take some books and loaf about his room, going to bed early and sleeping until he wakes up in the morning. The motor car is best left at home on the week-end rests and on vacations as well. It is much better to sit back in a comfortable Pullman seat and relax than to be under the constant tension of driving.

Rest before and after meals is an important part of the regimen. After breakfast he should rest before starting out on his daily duties. At noon he should rest before his lunch and after lunch as well. In the evening the patient should get home in time to become thoroughly rested before dinner and should rest after dinner for an hour or more. Fatigue interferes with digestion in adults as well as in children. After meals, during the period of digestion, the heart and circulatory system already have an additional load.

The diet should be adapted to the needs of the individual patient. It should be such as to be readily digestible and one which cannot cause gas. Full, heavy meals are to be avoided. In some patients hydrochloric acid with the meals is indicated and may help in preventing attacks.

There is no indication for any restriction of diet and certainly there is no need for restricting the protein intake, as is

occasionally done It should not be below the daily protein needs In the overobese an appropriate diet should be ordered It is better if the diet is adjusted to the point so that it just about equals the patient's caloric needs In some cases it may be better to prescribe small meals with the addition of something between meals, if necessary The diet should afford a carbohydrate ratio sufficient to maintain the blood sugar at the higher limits of normal If diabetes is present, it should be controlled without insulin in those cases where it is possible to do so and still maintain an adequate diet In the cases where insulin is necessary, as it frequently is, the patient must adhere to a known and prescribed diet and to a prescribed insulin dosage The insulin always should be such as to maintain the blood sugar at a level at which hypoglycemia can be certainly avoided It is better to allow a trace of sugar to show in the urine

Occasionally attacks of angina pectoris occur in those who are suffering from symptoms of duodenal ulcer Some of these patients have an active ulcer with deformity of the bulb Others do not show an actual deformity but show evidence of an overirritable bulb with frequently no ulcer Such patients should be put under ulcer management and especially ulcer management associated with the use of some mild sedative as phenobarbital In many of these cases the symptoms are only another expression of Dr Roberts' "spasmogenic aptitude"

Many angina patients have a constipation of the spastic type This should be controlled as far as possible by diet A glass or two of hot water on arising may help If necessary, liquid or solid vaseline or one of the preparations of vaseline which do not contain a cathartic can be used Solid white vaseline is especially valuable It is not hard to take A large ball of it can be placed on the back of the tongue and swallowed night and morning If the diet does not afford sufficient bulk, agar can be added

If cathartics are necessary, the milder saline laxatives are

preferable Strong or stimulating cathartics may predispose to attacks, especially in those patients with spastic colons

The question of the use of liquor frequently arises Some patients are unquestionably the better for its moderate use, especially the more elderly patients Others with even a very moderate or small amount may have gastric disturbance which predisposes to an attack. Liquor is best avoided as a general rule

Just what to decide in regard to the use of tobacco must be decided according to the individual patient. Its use should certainly be moderate and it should not be used on an empty stomach If there is any question as to its harmful effects, it should be omitted

Such general directions as have been indicated above are quite as important as any part of treatment. A little pocket booklet with some very sensible and wise general directions for the patient's use has been prepared by Dr John Sproull of Haverhill, Massachusetts, and is a very good guide for the patient to have

When possible, the patient should spend the winter in a warm equable climate High altitudes are always to be avoided and especially so where there is much actual cardiac pathology For every increment of elevation, the heart has just so much more work to do Patients will frequently return from a vacation at a high altitude and inform you that your warnings in that regard were quite unnecessary, but others will return and tell you that they wish that they had obeyed your directions The same caution applies in regard to trips by plane Conditions may force the plane to remain at a high altitude for a long period A trip from Omaha to Chicago at 6000 feet undid the results of months of care in the case of one patient.

The attacks themselves are best relieved by amyl nitrite or nitroglycerin Amyl nitrite acts a little more promptly but is less convenient and always conspicuous, as well as being a source of annoyance to others Nitroglycerin dissolved under the tongue acts promptly enough for all clinical pur-



poses It is especially useful because it can be readily carried in the pocket or handbag and can be used without attracting attention at any time It loses strength rapidly when exposed or with age, it is best purchased in the small hypodermic tube of 20 tablets and kept tightly stoppered The usual dose is  $\frac{1}{100}$  grain There are patients who have uncomfortable symptoms or very rarely syncope with this dose and in whom a smaller dose does better The dose can be repeated if necessary The other nitrites act too slowly to be of much use, although their action is more prolonged If the attacks last long enough to demand a drug with more prolonged action, there are more serious aspects which must be considered

Heberden recommended "spirituous liquors" in the treatment of the attack and they are still valuable Hot, strong, black coffee is of help in some cases and may act as a substitute when other means are not available

A large part of the treatment between the attacks consists of the evaluation by the physician of just what it is that predisposes to the attack and precipitates it, and the directions which the physician gives the patient as to his care There is no remedy which will alter anatomical changes in the arteries and certainly none which will "cure" angina pectoris Nor is there any one drug which will afford any degree of relief in all cases But most cases can at least be materially helped by medication

In our experience here we have obtained better results by the use of the purine base group than with any other one medical treatment In some few cases almost complete relief has been obtained In most cases some degree of relief has been obtained, from a very great deal to moderate or slight As would be expected from the nature of the conditions which cause the pain in angina pectoris, there must be cases where relief cannot be obtained

It is not possible to say which of the purine base preparations is the most valuable On some occasions a result will be obtained with one preparation when another has not been

effective, or one will cause uncomfortable symptoms and another not. Of the theobromine preparations, we use most frequently theobromine calcium salicylate (theocalcine), or the alkaloid theobromine, and of the theophylline preparations, theophylline ethylene diamine (aminophylline) or theophylline calcium salicylate (phyllicin).

We make use of most of the other preparations at one time or another, however, partly because of their lower cost and partly because, for no reason which we can assign, occasionally one preparation seems to work better than another.

The alkaloid theobromine we prescribe in 5 and 7.5-grain tablets, and theobromine calcium salicylate (theocalcine) 7.5 grain tablets, one or two at a time. Theobromine sodium salicylate and theobromine sodium acetate we use in 10 grain capsules. The acetate salt carries a little more theobromine than does the salicylate. The dosage of the theophylline preparation is much lower in each case. The alkaloid theophylline is prescribed in 2 grain capsules, and the theophylline ethylene diamine (aminophylline) in 1.5 grain tablets, and the theophylline calcium salicylate (phyllicine) in 4 grain tablets.

All of the series have some disadvantage. Most of them may, and occasionally do, cause some unpleasant symptom in the way of nausea, gastric distress, headache, or nervousness. Theobromine calcium salicylate (theocalcine), because of its insolubility in the stomach, rarely causes unpleasant symptoms. The same is true of theophylline ethylene diamine (aminophylline). Theophylline calcium salicylate is equally effective and in some cases more so, and is usually well tolerated. All are best taken during the meal, a little food, then the drug and then the rest of the meal.

In experimental animals the theobromine preparations cause the greatest increase in coronary flow. The theophylline preparations are next, and caffeine the least effective. Theophylline ethylene diamine is quite as effective as a vasodilator of the coronaries as the theobromines because of the

vasodilator effect of ethylene diamine The theophylline calcium salicylate is quite as effective clinically in our experience

It is probable that some degree of tolerance is acquired of these drugs and Meyer has shown that not only tolerance is acquired, but a cross tolerance is acquired as well to others of the series We have assumed that such a tolerance is acquired clinically and have thought on some occasions that we have had evidence of this in some of our patients In order to avoid acquiring a tolerance, we have been in the habit of using the theobromine preparation one week and a theophylline preparation the alternate week The effects of this procedure is open to question where cross tolerance is so easily acquired With the same objective in mind, we have, where possible, used the medication for four days of each week and omitted medication for three days While a tolerance may be acquired there are certainly a great many patients who do not acquire such a tolerance

In the second case which we are to show this morning, theobromine preparations have been used steadily for seven years and for the past two years the patient has insisted upon using the alkaloid theobromine because she gets more relief with this preparation If she omits the medicine, the symptoms return as before

Another case returned for a check-up this week who has used the theobromine preparations steadily for eleven years and is still benefited

In order not to discourage the patient by any untoward effects, at the onset, we start with the theobromine calcium salicylate (theocalcin) which only very rarely causes distress, and then alternate with the theophylline ethylene diamine (aminophylline) or the theophylline calcium salicylate (phyllcin) Later we try other preparations and use the preparation and dosage which we find the most effective and the best tolerated

We think that in these patients who have shown little or no effect at first and then have done exceptionally well, there is an actual improvement in the underlying condition This

has been the opinion, also, of Dr F M Smith and he has confirmed this by his experimental work on the dog, showing that with the use of the purine base diuretics, there is an actual increase in the anastomoses and collateral circulation consequent upon the constant vasodilation produced by these drugs

Good results have been reported from the use of various tissue extracts. In experimental animals when administered into the vein, they have been shown to increase the coronary flow, and to dilate the peripheral vessels. In the preparations which we have used, we have observed little or no increase in the coronary flow in the experimental animal.

In peripheral vascular disease the clinical results following the intramuscular injection of tissue extract have been definitely good, although there has been some doubt as to whether this clinical improvement was due to a vasodilation or not. It is possible that the good results may be due to some factor other than vasodilatation and that they are concerned in some way with muscle metabolism.

In our experience with the use of tissue extracts, which has been relatively small, we have not found them as useful as agents of the purine base series, and we certainly do not feel that they could replace these drugs. In some cases we feel that they have been of very real value, and have caused further relief of symptoms when the purine base drugs were not wholly effective. In such cases we have continued the use of the purine base drugs. On a few occasions the patient has not done as well with their use, and we have considered that a fall in the systemic blood pressure was responsible for the result. Care must be taken not to inject into a vein.

Different nitrite preparations, especially those with a more prolonged action, as erythrol tetranitrite or sodium nitrate, may be used routinely to prevent the recurrence of attacks. In our experience they have been rarely necessary, but there are occasions when they should be given a trial. They should not be used to the point of maintaining a lowered blood pressure and in some cases we think that we have seen untoward

results from their daily and frequent use. The nitrite preparations are occasionally given to a patient in order to undertake some additional effort without an attack. We do not recommend this to our patients and prefer that they do not undertake the additional effort.

Alcohol taken wisely and moderately has a place in the treatment of angina pectoris in certain cases. "Wine and cordials taken at going to bed will prevent or weaken the night fits," wrote Heberden, and it is a method worth trying in some cases. Heberden goes on to state that "nothing does this as effectively as opiates" and they also must be considered today, and especially so when age or a degree of illness is reached when habit forming is not to be feared.

Phenobarbital is of great value, especially when used in conjunction with the purine base drugs, or with other medication. We prefer to use it separately and not combined with theobromine, in order that we may vary the dosage as necessary. We attempt to use a dose which will produce a sedative effect without drowsiness. Sometimes one of the more rapidly acting derivatives is of more value at night.

Potassium iodide is used a great deal as an orthodox method of treatment. I have never been able to persuade myself that I could attribute any favorable results to its use. It certainly will not bring about any anatomical changes in the vessels. But where a drug has been used so consistently for so long a period, one wonders if there is not some possible virtue in it after all.

Digitalis is best not used at all except in the cases where there are definite indications for its use, as auricular fibrillation or a passive congestion which does not yield to other measures. There is no reason in ordinary cases why it should be used, and there are reasons why it should not be. We have obtained definite experimental evidence that digitalis decreases the coronary flow and clinically attacks have been made more severe and more frequent by its use or have been precipitated by its use. Especially should it be avoided where there is an aortic insufficiency. In aortic insufficiency, the coronary flow

is at a great disadvantage because of the low diastolic pressure. In most cases this is compensated for in part by an increase in the systolic pressure and by an increased pulse rate. Digitalis may lower the systolic pressure, may decrease the pulse rate, and possibly further lower the diastolic pressure, each of which factors would tend to cause the coronary flow to become inadequate.

Those cases in which pernicious anemia is a factor influencing the attacks yield to proper treatment. If a hypoglycemia forms the basis for the attacks, diet or glucose by mouth should be sufficient.

Various surgical methods have been devised for the relief or prevention of anginal attacks. An adequate discussion would take more time than we can give here. The operations involving ganglionectomy or sympathectomy are applicable only in a few chosen cases and the results have not been encouraging. Paravertebral injection of alcohol into the first five thoracic ganglia, as advocated by White, is more effective and does not carry the mortality that the more extensive surgical procedures do. More recently Blumgart and his associates have advocated total thyroidectomy in the treatment of angina pectoris. Their results as observed over a period of two or three years have been satisfactory and our own very small series has shown good results, the cases must be chosen with great care and it is not a procedure to be used indiscriminately.

I have postponed showing cases until the last, and we shall be obliged to show them very briefly and give only the essential history and findings.

**Case I.**—This first patient is a white clerk, aged fifty four who entered St. Luke's Hospital in September 1929 with typical attacks of anginal pain. These attacks came on while walking from his home to the elevated railroad and from the train to his work, and again on going home in the evening. He would have 1 or 2 attacks in the 4 blocks which would necessitate his stopping and sitting down. They would wear off in a minute or two and he would proceed. For several months the attacks would appear only once or twice a week, but for two weeks before entrance there were frequent daily attacks. Physical examination revealed no evidence of cardiac pathology. A 2 meter chest plate showed a heart of normal contour whose transverse diameter was

43 per cent of the transverse diameter of the chest. The great vessels were normal. An electrocardiogram shows the T wave in lead II leading off from very slightly below the iso-electric line. Otherwise it was normal. Blood, blood chemistry, Wassermann and urine were normal. Blood pressure varied around 120 systolic and 80 diastolic.

The patient was discharged on the third day. He was instructed to take theocaine, 7.5 grains four times a day for one week, and metaphyllin, 1.5 grains, each alternate week. There was no improvement and October 13, 1929 alkaloid theobromine, 7.5 grains, was substituted for the theocaine, giving about twice the dosage of theobromine. November 11th the pains were some better, and definitely better on December 22nd. February 9th he reported that the attacks were "about gone" and were better in the week when he took the alkaloid theobromine. March 9th he reported that he had had no attacks during the preceding week. There was no pain until October when there was some slight pain, but not what he would call an attack. There was no pain until July 1, 1932 when he had some pain again. On both of these occasions no history of any provocative cause could be elicited. There have been no attacks since that time. Beginning in 1933 the medication has been taken only intermittently for a month or two at a time and a month without medication.

It does not seem probable that there was a coronary thrombosis at the onset. There was no history of an attack, but there was a history of isolated short attacks of anginal pain becoming more frequent. It is not probable that anastomoses and collateral circulation improved spontaneously in so short a time. We have, too, the definite statement that he was better the weeks that he was taking the more effective dose of theobromine, than when on the relatively smaller dose of metaphyllin.

We consider that in this case relief was obtained by the medication and that the long-continued medication and continuous vasodilatation increased the anastomosis and collateral circulation, resulting in the present complete relief from symptoms.

**Case II**—This next case represents a very much less favorable type for treatment, but is a case which we are very sure has been benefited by treatment.

The patient is a housewife, aged sixty-two. She entered St. Luke's Hospital on April 18, 1928, complaining of dyspnea upon exertion and edema of the lower extremities which had been present for three years, and of attacks of anginal pain, present for the past two years. The attacks of angina pectoris occurred several times daily, especially after meals. There were also nocturnal attacks.

Except for the obvious evidence of aortic insufficiency she is a normal appearing woman for her age and was not essentially different at her entrance seven years ago.

At the time of entrance physical examination revealed a very typical aortic insufficiency with a loud diastolic and a loud systolic murmur over the aorta and an associated mitral insufficiency. There was nothing in the previous history to indicate the occurrence of rheumatic fever or syphilis. There have been no pregnancies.

A 2 meter chest plate showed a left ventricular hypertrophy, the left border 11.7 cm. to the left and the right border 3.1 cm. to the right of the mid-sternal line. The transverse diameter of the heart occupied 55 per cent of the transverse diameter of the chest. At the present it occupies 56 per cent of the transverse diameter of the chest. Repeated Wassermann and Kahn tests were negative at this time and have continued negative. Blood chemistry was normal throughout. Routine blood examinations showed only a moderate secondary anemia. The urine was normal except for a trace of albumin. The electrocardiogram showed evidences of extensive coronary and myocardial changes.

The blood pressure on entrance was 310 systolic and 50 diastolic. It has continued high and since leaving the hospital has varied from 300 to 198 systolic and 56 to 44 diastolic. It has averaged high in January and less in July. It apparently bears no relation to the pain.

The patient remained in the hospital for two weeks. During this period of bed rest the evidences of passive congestion disappeared. The attacks of anginal pain were less frequent than when up and about, but the patient still continued to have several attacks daily. On discharge, she returned to the follow-up clinic, where she was placed upon the purine base diuretics at once with immediate improvement. She had less pain when up and about with this medication than when at rest without it. During the intervening seven years she has continued for the most part upon this medication. It has been discontinued several times for periods of two to four weeks. During these periods everything at all reasonable that we have heard of has been tried. Nothing was found which would give her as much relief as her regular treatment. During the last two years she has been upon the alkaloid theobromine and theophyllin ethylene diamine. Attacks still occur but are less frequent and less severe. There will be periods of one to three weeks when there are no attacks, and at other times there will be 1 or 2 or 3 attacks a week. The last few months there has been less limitation of activity and fewer attacks than at any other time. Moderate shortness of breath still persists but there is no edema.

During these seven years of observation it is possible that part of the improvement has been due to the increased anastomoses and increased collateral circulation which might have occurred spontaneously with time. The improvement, however, began at once with the administration of the purine base



drugs. The symptoms even now recur with greater frequency and severity if the medication is discontinued. She is better also when upon the alkaloid theobromine than when trying the other preparations. We feel that the medication has played a part in producing the increase in the anastomoses and collateral circulation which has probably occurred.

**Case III**—This especially healthy appearing man represents an unusually favorable type for thyroidectomy for the relief of anginal pain and has shown especially good results.

The patient is an electrician and the proprietor of a radio sales and repair shop, aged fifty-nine. He came to the out-patient department November 12, 1933, complaining of frequent attacks of anginal pain, loss of weight, and sleeplessness present since the previous June. The attacks of pain occurred frequently during the day on slight exertion, and at night while lying in bed he had as many as 12 attacks. He was unable to work and had been practically bedfast.

There was a very obvious toxic thyroid present, with a metabolic rate of plus 32. The blood pressure was 164 systolic and 90 diastolic with a pulse of 112. Although he improved during his preoperative course of iodine and there was an amelioration of his thyroid symptoms, there was no change in the frequency or the severity of his anginal attacks. Theobromine sodium acetate, 40 grains daily, showed no effect.

He entered the hospital on December 3, 1934 and a subtotal thyroidectomy was done the next day by Dr. S. W. McArthur. After an uneventful post-operative recovery, he was discharged on the eighth day.

He has had no attack at any time since the operation. The night before the operation he had several attacks of pain. Even changing his position or sitting up might bring on an attack. The night following his operation he got out of bed and closed the window and moved a screen without pain. It would seem that there was something else in addition to the lowered metabolic rate which was responsible for the absence of pain after thyroidectomy. It is very improbable that the rate was materially lowered in the few hours intervening between the operation and his undertaking exertion that he could not possibly have undertaken before the operation. On the ninth day after the operation the rate was plus 25.3, later falling to minus 25.6, where it still remained last month.

He regained his weight rapidly, passing his former weight, and returning to his usual weight when he resumed exercise. There have been no attacks between the time of operation and now. When I called him to ask him to come to the hospital, he had just returned from a 2-mile walk on a cold winter evening. There is no shortness of breath. He has even experimented with running.

This case has been, of course, an especially favorable case. The thyroidectomy would have had to be done, irrespective

of the angina pectoris. The case does not belong with the group which has been reported from Boston and which has shown such favorable results.

In showing this second case of thyroidectomy done for the relief of anginal pain, I do not wish to be understood as advocating this line of treatment except in a very few chosen cases. In the other case just shown, his condition quite aside from the angina indicated a thyroidectomy. In this case which we are about to show, every other possibility was exhausted before a thyroidectomy was done. Most cases can be handled medically and we think, as far as we know now, to better advantage. Until cases have been followed for a long period of time and the results watched carefully and impartially, nothing definite can be said as to the advantages of thyroidectomy in chosen cases of angina pectoris. It is obvious that no matter how satisfactory the results may be in some cases, the procedure will never be applicable to more than a well chosen few.

In this second case of thyroidectomy, the operation was only done after months of unsuccessful efforts with other treatment. We were especially hesitant because of the associated pathological conditions.

**Case IV**—The patient is a normal appearing man of fifty three, a real estate salesman. He entered St. Luke's Hospital in September 1933 because of attacks of angina pectoris. Until two months before entrance the patient was in his usual health. About this time he began to have occasional attacks of anginal pain. These rather rapidly became more frequent and more severe until it became impossible for him to continue at work. Attacks are now precipitated by the slightest exertion so that he is practically bedfast. There are one to several attacks at night. There has been no shortness of breath but there is some mild substernal pain present between the attacks part of the time.

Physical examination showed nothing of great significance. The heart was within normal limits of size. There was a soft systolic murmur at the base, under the sternum not transmitted. The heart sounds and rhythm were normal. Physical examination at present is quite unchanged except that he has gained weight and has not the nervous apprehensive appearance which he had at that time.

Laboratory examinations showed the heart to occupy 4.7 per cent of the transverse diameter of the chest, 8.7 cm. to the left and 4.7 cm. to the right of the midline.

The electrocardiogram showed only moderate variation from normal between the attacks, but during the attacks the tracing showed a marked deviation from normal type, indicative of coronary involvement.

The blood pressure was 120 systolic and 74 diastolic on entrance, and has since varied only slightly above and below these levels.

The blood Wassermann and Kahn were strongly positive, and the spinal fluid Wassermann was mildly positive. There was a low paretic curve with the colloidal gold test. The basal metabolism rate was minus 12. Routine blood examination showed a moderate secondary anemia. The urine was normal upon examination and functional tests were normal.

The patient remained in the hospital for seventy-one days, and then was discharged to a convalescent home. During this time he received various theobromine and theophyllin preparations. Neither these drugs nor other attempts at medication showed any definite effect. He improved enough so that he could be up and around the ward, and while he was much better, still had daily attacks and occasionally attacks during the night. There was no improvement that could not be accounted for by bed rest and time.

We feel that in cases of syphilis involving the circulatory system we should be very careful in regard to our antisyphilitic treatment. What we do depends entirely upon the cardiac condition and treatment directed at the syphilis may be omitted altogether until the cardiac condition has improved. We are very certain that we have seen even mild cardiac conditions become rapidly very much worse with vigorous or even mild arsenical therapy. As Dr. William Allen Pusey puts it, "Syphilitic tissue is better than no tissue at all." There are doubtless cases where vigorous arsenical treatment does not show untoward results, but we prefer not to take the risk. We start with mercury or mercury and iodide, and with bismuth in alternate courses, and even watch these closely. If subjective symptoms are aggravated or the pulse rate is increased, we stop for a time.

In this case symptoms became definitely more pronounced with mercury and with mercury and iodides or with bismuth, and all antisyphilitic treatment was discontinued temporarily.

After discharge the patient continued to return to the outpatient department. Mercury or bismuth were again tried at various times for short intervals, but could not be continued steadily. The purine base drugs in one form or another were

continued. He felt that he was better upon the alkaloid theobromine and he received this most of the time.

In June, 1934, he was a little better clinically, but he still had several attacks daily and could not return to his usual occupations. The convalescent home could not keep him longer, and he could not take care of himself alone in a rooming house. It was decided to do a thyroidectomy in spite of the unfavorable outlook. He was accordingly operated upon June 30, 1934, by Dr. H. E. Mock, who did a subtotal thyroidectomy. He made an uneventful recovery and was discharged on the twelfth day. There were no anginal attacks after the operation during his stay in the hospital.

Since his discharge he has been free from his former attacks, except for a few scattered attacks on prolonged effort. He has some mild pain, described as a burning sensation, under the upper third of the sternum on walking. This may come on in two blocks or it may be only after seven or eight blocks and it goes away as soon as he stops.

He is taking the alkaloid theobromine and theophylline calcium salicylate on alternate weeks, as he states that he feels better on these two.

It is interesting that since the thyroidectomy the metabolic rate has been higher than before, and has varied between minus 6 and minus 8.

He is receiving alternate courses of mercury and bismuth in the skin clinic. At first these were discontinued occasionally because of some increase in the substernal pain, but now are going along continuously. Because of the spinal fluid findings we do not wish to lose any more time than is necessary in our antisyphilitic treatment and will begin to use the arsenicals sooner than we otherwise would.

He is still limited in his activities, but has returned to work and to his normal mode of life. Even with the residual substernal pain, which was to be expected, he is infinitely better off than with the recurring and frequent attacks of angina.



## CLINIC OF DR LOWELL D SNORF

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#### PAIN IN THE ABDOMEN CLINICAL SIGNIFICANCE AND CONSIDERATION OF RELIEF

PAIN as a symptom of abdominal pathology is deserving of careful study since it is one of the most important single symptoms and often most dramatic to the patient. In consideration of this symptom we must use it in the broadest sense, varying from distress of mild character to that of a severe agonizing colic. This interpretation is obviously necessary since vague distress at times may develop into acute pain and also because it will necessarily vary with the individual reaction. Sensations of pain may be materially altered by the apprehensiveness of the individual. A practical analysis of pain should contemplate its origin and some rationale for its particular characteristics, some understanding of the pathologic physiology involved. One must consider, furthermore, whether the pain is intra abdominal or extra abdominal, as the symptoms may be very confusing.

In the interpretation of pain we must of necessity relate it to other associated symptoms. Seldom will it occur that a definite pattern of pain will be adequate in any instance to make a definite diagnosis.

Generally speaking, the walls of the abdominal cavity are innervated by the cerebrospinal nerves, while the viscera are innervated by the splanchnics. The lower surface of the diaphragm is supplied by the phrenic, the afferent nerves having their origin in the fourth cervical segment of the cord. The anterior and lateral walls of the abdomen are supplied by the lower six thoracic and first lumbar nerves. Pains originating in the stomach or other viscera may be referred to the surface

of the body supplied by the corresponding spinal segment. It is obvious, therefore, that diseases of the spinal cord may refer pain from these corresponding root segments which may simulate disease of the several viscera. Gastric and intestinal pains are thought to be due either to increased tension of the muscle wall, to involvement of the peritoneum or both.

Rapid distention of the stomach has been observed to produce pain, the intragastric pressure indicating that the muscular tension was the chief factor involved. The spasm of the pylorus is another example. There is a close relationship between hypersensitivity of the gastric nerves and pain as exhibited in the distress of ulcer of the stomach or duodenum. The absence of pain during observation of the large visible peristaltic waves strong enough to lift the abdominal wall or similarly noted during fluoroscopic study would indicate that the ordinary conception of increased muscular tone was not the prime factor causing the pain.

A peculiar conditioning of nervous reflexes associated with an increased sensitivity of the nerves of the stomach or duodenum accounts best for the pain activated by the acid secretion.

The mucous membrane of the stomach and intestines is relatively insensitive to thermic, tactile and chemical stimuli. When weak acid solution is put in the stomach bearing an active ulcer, pain will be produced and if, as Palmer has shown, the ulcer is quiescent, no pain will result. This would indicate that acid is an essential factor in the production of pain when proper conditioning of nerves occur.

It is more difficult, however, to produce pain in the intestines. Sudden distention of the colon from gas associated with excessive peristalsis or the injection of large quantities of water in the colon while in an irritable state, will often produce marked pain. These observations are essential to bear in mind when opportunity presents itself in test-out in the differential diagnosis between ulcer and disorders in the colon.

The peritoneum covering the stomach and intestine is also relatively insensitive, whereas the parietal peritoneum is very sensitive. It seems quite reasonable, therefore, that tension

on the parietal peritoneum from distention of the viscera produces much the greater part of pain. This is particularly true in perforations, local peritonitis and intestinal obstruction. The cramplike pain of intestinal obstruction seems to be due to the extreme tone initiated by excessive and vigorous peristalsis. When free portions of the intestine are involved, there is a tendency for the pain to be referred to the epigastrium or the neighborhood of the umbilicus, whereas that arising from fixed portions is usually related directly to the region involved. Let us now analyze the pain picture as it is related to various disorders in the abdomen.

A typical colic frequently means a definite pattern of pain or distress. We must also ascertain whether the pain is acute or chronic. An acute pain usually means sudden onset from infection, perforation, obstruction, or some intra abdominal accident, as vascular occlusion.

Inquire as to associated temperature, shock, nausea, or vomiting. In the chronic recurrent pain picture is there relationship to food ingestion, either of quality or time, and does bowel movement or certain physical activity influence the pain? Whether acute or chronic we must attempt to determine the point of maximum intensity of pain, the direction and point of reference. Is it influenced by any test procedure and with what other symptoms does it occur?

Before discussing the intra abdominal lesions, let us analyze briefly the pain as it may be referred to the abdomen from extra abdominal sources.

Diaphragmatic pleurisy or pneumonia of the right lower lobe may produce a referred pain to the upper abdomen or even as low as McBurney's point. If jaundice is an accompanying symptom, as is not infrequently the case in pneumonia, the deception is complete. Only recently we observed a patient who had had a previous x ray and diagnosis of cholelithiasis, who at the time was suffering from very severe upper right quadrant pain. Two days passed before there were sufficient physical signs in the lower right chest to bear out the diagnosis of pneumonia. The pain in the upper right



quadrant, although severe, was at no time associated with rigidity of the right rectus muscle and the temperature was out of proportion to the abdominal findings. Pneumonia was finally positively diagnosed.

Pulmonary tuberculosis or bronchiectasis may simulate a lesion of the abdomen. Tuberculosis particularly will be confused and rightly so, since it is not infrequent that we find intestinal tuberculosis following the pulmonary involvement, yet the involvement of the diaphragm, with pain referred to the neck through the phrenic nerve and a negative gastrointestinal study, will make the diagnosis certain of uncomplicated pulmonary tuberculosis.

The abdominal nerves may be affected at their source, as in tabes dorsalis, osteo-arthritis, Pott's disease or osteomyelitis. When the bodies of the vertebrae are involved, it may require no little ingenuity to prove the origin of the pain. Pressure over the involved bone may reveal marked tenderness, as in Pott's disease or osteomyelitis. Bed rest and a body cast may be finally necessary to diagnose correctly the bone involvement.

A coronary accident may so closely simulate an intra-abdominal disorder as to lead to serious consequences. A brief case report of such an experience might be worth analyzing. A man, aged sixty-five years, came to the hospital complaining of severe right upper abdominal pain. He gave a history of similar attacks over a period of several years. As a rule, these attacks were sufficiently severe to require morphine for relief. At no time did this man note pain over the precordium, much less a reference pain into the left shoulder or left arm. He showed no particular evidence of shock, blood pressure was down to 100 systolic. Previous to my examination he had had a Graham-Cole test which failed to visualize the gallbladder. Diagnosis, therefore, of gallbladder disease seemed probable. However, when I was called to see him, it was quite evident that the man was having some distress related to the cardiovascular system. His chief complaint on the morning of my examination was that of dyspnea.

and with continued observation it became apparent that the dyspnea was associated with Cheyne Stokes respiration. The cardiogram was taken at once and revealed marked evidence of myocarditis and disturbance in the conduction time. He succumbed to this coronary accident, and postmortem revealed a large infarct in the myocardium. The gallbladder was normal. Evidences of numerous healed infarcts were found in the myocardium, probably accounting for the previous gall bladder like colics that he had experienced before.

Experiences of this kind are quite common. Many physicians have noted similar confusion ever since Herrick called our attention to coronary occlusion in 1912, but one is never so impressed by the possibilities of confusion until he has made a number of mistakes. Recently a not dissimilar problem was presented. Acute pain in the abdomen was thought to be due to a perforated duodenal ulcer. The patient gave a history of a duodenal ulcer of many years' standing and had taken rather poor care of himself. Shortly before the onset of this acute attack of pain, he had noted definite recurrence of his ulcer symptoms. He experienced a sudden epigastric pain radiating slightly upward into the chest. This radiation, however, was rather diffuse and had none of the typical earmarks of cardiac accident. His temperature ran between 99° and 100° F, pulse slightly increased, blood pressure from 95 to 100 systolic, and no abnormal findings in the chest were noted. Because of the previous history and the present localized pain in the abdomen, it was thought he had a perforating ulcer of the forme fruste type and he was operated upon. No perforation was found, but the scar of the old duodenal ulcer was quite easily visualized. The following day definite evidences of pericarditis were noted. Recurrence of pain was also observed, and after several weeks the patient made a relatively uninterrupted recovery.

I think it is important that this mistake in diagnosis be commented upon inasmuch as the patient was subjected to a very serious surgical procedure that might have resulted in disaster. Following his operation a cardiogram was taken

and presented conclusive evidence that we were dealing with coronary occlusion. The mistake in the diagnosis might have been obviated had we taken a cardiogram prior to the operation. Frequently, of course, the cardiogram does not reveal any positive evidence within the first twenty-four hours and, therefore, in the presence of a negative cardiogram we must always be extremely cautious in the diagnosis of upper abdominal lesions in an individual whose age is compatible with coronary accident.

Pains from intra-abdominal lesions may best be discussed from the standpoint of the organs involved, especially when the pain pattern is fairly exact.

A typical attack of biliary colic is characterized by severe pain in the right upper quadrant of the abdomen radiating through to the back and usually to the right shoulder blade. The paroxysm usually begins and terminates abruptly. Very commonly there will be a residual soreness over the region of the gallbladder for several days. According to the criteria stated above, for the consideration of pain, we have here a definite point of maximum intensity under the right costal margin and radiation to the right shoulder blade. It cannot, however, be related to a definite physiologic functional disturbance since it seldom occurs regularly following meal taking. It is, however, often precipitated by an excessively heavy meal, particularly one containing fat. There is also associated symptoms of nausea, sometimes vomiting, and an increase in temperature. When jaundice occurs, the diagnosis is much more complete. This typical attack is usually connected with the passing of a gallstone. When obstruction of the cystic or common duct occurs associated with an active infection in the gallbladder, then the accompanying symptoms of shock, high fever and continued pain are evidences of empyema of the gallbladder. A sudden onset of pain with marked rigidity, evidences of shock and infection make the diagnosis more difficult because then perforated ulcer, acute hemorrhagic pancreatitis, coronary occlusion or mesenteric thrombosis must be considered.

Renal colic very frequently presents a typical picture. The colic may be due to kink of the ureter or the passage of a stone or of a stone blocking the kidney pelvis at the uretero-pelvic junction. This colic is a paroxysm of pain noted in the flank and frequently radiating downward toward the bladder and into the thigh or into the testicle, penis or labia on the side involved. This pain traverses the field of distribution of the eleventh thoracic to second lumbar spinal segments. Burning and frequency of urination are quite constant. The finding of red cells in the urine is an added diagnostic point. We have observed that distention of the kidney pelvis by retrograde pyelography produces a typical colic. The hydronephrotic kidney of long standing causes little distress unless an obstruction of the ureter, as in Dietl's crisis, occurs. The pain from stone or kinking of the ureter is due undoubtedly to an increased tone and distention of the kidney pelvis or of the ureter itself rather than to direct irritation from the stone on the mucosa. Less typical attacks of pain occur from disturbance of the kidney and require a great deal of care in the differentiation from the more chronic type of distress produced by the colon or by recurrent appendiceal infection.

A woman thirty two years of age, complained of pain in the right lower quadrant in the region of McBurney's point for several years. Because of this constant annoying pain and the associated disorder of the colon function the appendix was removed. The distress originally complained of continued. There was never any sign of acute colic, no frequency or urgency of urination and no abnormal urine findings. At the time of our examination it was noted that the kidney was easily movable and when placed upon her left side the kidney traveled out to the region of the umbilicus. She was then examined while in the upright position. The kidney was found to be at the level of the crest of the ilium and when pulled downward it was possible to reproduce the pain which she had previously experienced. On advice her kidney was studied both by intravenous urography and retrograde pyelography. A marked ptosis with definite hydronephrosis and associated ureteral angulation were found. Proper measures were instituted and the pain disappeared at once.

I am quite convinced that every patient with chronic recurrent right-sided pain should be studied from the stand

point of possible kidney involvement Undoubtedly strictures of the ureter will produce such symptoms

An uncomplicated peptic ulcer usually produces a typical pain pattern Here it is extremely important to study the type of pain with relation to the several criteria noted above because in so doing we are able to evaluate to a great extent the nature of the lesion and something of the progressive pathology if and when such changes occur Distress of ulcer, as we are all quite familiar, may be no more than fulness or present the severe agonizing pain associated with acute spasm or perforation The pain of uncomplicated ulcer, whether gastric or duodenal, is usually noted in the midepigastrium It seldom appears in the morning before breakfast, usually occurs an hour or two after meals and occasionally it noted at twelve to two o'clock in the morning It regularly is relieved by adequate alkali, food, vomiting and aspiration

When the ulcer becomes complicated by obstruction, perigastritis or perforation, the pain then may vary considerably In the presence of obstruction it will frequently continue until the next meal When it is associated with obstruction due to spasm, the pain frequently will be quite intense, but will still be relieved by food and alkali When obstruction is associated with definite cicatrix formation, a continued secretion is likely to be present and food and alkali may not give such complete relief Aspiration or vomiting, however, invariably give prompt and complete relief Perigastritis, or periduodenitis, indicates an inflammation of the peritoneum and here the pain is not always completely relieved by food and alkali, in fact, sometimes it is aggravated Furthermore, the pain is likely to be accentuated by change of position and also by increased peristalsis

It is well to remember particularly this fact, that pain of an uncomplicated ulcer, whatever its cause, will be relieved by food, alkali, vomiting and aspiration If we will use this as a simple procedure for test-out observations, we will usually be able to differentiate very satisfactorily between gallbladder disease and peptic ulcer The following case report will sug-

gest the varying pathology in ulcer as indicated by the changing pain picture

A man aged forty came to the hospital seeking treatment for "ulcer of the stomach." He stated that he had had symptoms of stomach trouble during most of his adult life. As a young man he had what was called heart burn, occurring several days in succession, always appearing an hour or more after meals and relieved by soda. For this his appendix was removed. Within a few months he had a recurrence of distress that had more the nature of definite pain. This also was present an hour or more after meals. Periods of relief followed by periods of definite pain continued for more than ten years. The pain at first was relieved by food and soda and seldom appeared at night time. Later on however the pain was not so completely relieved by the average dose of soda. He was awakened frequently at night time and finally on occasion would vomit a large quantity of fluid out of proportion to the amount that he had taken. Vomiting would give complete relief of distress. One night he was suddenly seized with severe pain in his upper abdomen requiring the services of a physician who gave him morphine for relief. He states that the abdomen was extremely sore and tender for several weeks after this attack. He was forced to remain in bed because of the severity of the symptoms for more than a month. Some months after this particular incident he had a recurrence of symptoms similar to those experienced before the abdominal accident. He then for the first time in this long experience of gastric invalidism was given the advantage of an x ray study. It was found that he had a duodenal ulcer. He was operated upon and a gastro-enterostomy performed. The surgeon stated that the duodenum was immobile being fixed to the surrounding viscera by dense thick adhesions. His recovery from the immediate effects of the operation was uneventful. Some weeks later he experienced pains in his abdomen located to the left of the midline. This pain when it occurred would radiate downward to the groin. Occasionally he would vomit. He obtained relief from the distress by vomiting and by the taking of alkali. In fact, he obtained complete relief for a time on a carefully supervised diet. Subsequent examination of the gastro-intestinal tract revealed the stomach emptying through the gastro-enterostomy stoma and evidence of spasm in the stoma with distinct pain over this area. He was operated upon again and a jejunal ulcer resected.

Study of this patient reveals variations in the character of the pain based upon the changes in the pathology that developed from time to time. At first the pain and distress were that of an uncomplicated ulcer. Later the pain picture changed with the presence of obstruction. Later still the acute attack of pain was definitely that of a perforating ulcer of the duodenum and finally the pain produced by the complication from the perforating jejunal ulcer. When the pain of

ulcer shifts in its position from the midline to the left, one is usually dealing with a penetrating gastric ulcer or, more likely still, a jejunal ulcer. We have noted, as have others, that pain of a penetrating or perforating jejunal ulcer frequently radiates downward to the groin and, as Dr Ralph Brown reported, actual radiation into the left testicle. When pain is felt in the back, it usually means that the simple ulcer has become complicated by perigastric or periduodenal adhesions or that there has occurred a forme fruste type of perforation. Relatively acute pain recurring regularly day after day usually means spasm of the pylorus, while gradual decreasing severity of pain over a period of years suggests a scarring of the ulcer area. There is definite prognostic value in the proper and careful study of pain.

Abdominal pain is one of the commonest complaints, often the chief symptom, of the gynecological patient. It is usually low on one or both sides, less likely in the midline and seldom above the umbilicus. Pain of tubal and ovarian lesions is noted regularly over the brim of the pelvis and frequently on the corresponding side of the diseased organ. It is frequently precipitated by the onset of the menstrual flow. Repeated torsion of small ovarian cysts may frequently account for recurrent low abdominal pain. When the pain is generalized over the lower abdomen, it suggests the possibility of tuberculous peritonitis. Ectopic pregnancy and pyosalpinx are frequently confused with appendicitis. A pelvic examination may be the only means of definitely differentiating the conditions. On the other hand, sigmoid spasm, if prolonged and if of unusual severity, may be extremely confusing.

Appendicitis is invariably associated with some degree of pain. We are accustomed to accept the diagnosis of appendicitis on the basis of generalized abdominal pain, perhaps accentuation in the epigastrium and then gradually localizing in the region of McBurney's point with an associated local rigidity. The pain may be extremely severe, although as a rule it is accentuated chiefly by pressure directly over the cecum. The appendix alone is involved when the pain

is referred to the epigastric or umbilical region and later on involves the parietal peritoneum when the symptoms localize in the right lower quadrant. The pain of acute appendicitis may be confused with gallbladder or kidney disease, especially when the organ is located retroceally. Likewise, it may be confused with disease of the ovary or tube and particularly in older individuals difficult to differentiate from diverticulitis. When the pain is less severe or when it is associated with disturbances of the colon, particularly with the irritable bowel dysfunction, diagnosis may be very difficult indeed and it becomes obvious that the associated signs and symptoms of appendicitis must be taken into account rather than relying entirely upon the pain picture.

The irritable and spastic colon may produce pain which will mimic the pain of peptic ulcer, gallbladder colic, appendicitis, intestinal obstruction, or pelvic disorders. The pain usually shifts from one section of the abdomen to the other, may be aggravated by food ingestion, cold drink, chilling the surface of the body and often precede a bowel movement. It is induced by or aggravated temporarily by a large water enema. Coarse foods usually offend it. It may be associated with extra gastro-intestinal pathology, as in thyrotoxicosis and Addison's disease. It is frequently influenced by infection of the gallbladder, appendix, and by irritating lesions about the rectum and anus. It may be confused with lead poisoning, diverticulitis and postoperative adhesions. The possibility of an allergic state must be always borne in mind.

The colon is frequently palpable throughout its entire course and the patient is often quite conscious of the segments involved. The pain may subside and be followed by a sensitiveness and unrest comparable to pain, but of a less severe degree. In long continued irritabilities with or without ulcerations or inflammations there is frequently an associated psychic disturbance which requires the most careful evaluation in order to obtain a successful cure. The more often we meet these particular intestinal invalids, the more we appreciate the factor of mental instability as one most necessary



to treat Mucous colitis, although manifesting primarily a colon disorder, is invariably associated with a definite nervous makeup. The pain associated with expulsion of mucus is, I think, no different than spasm of the colon in the non-inflammatory colon, and mucous colitis is in reality a highly irritable colon with excess mucous secretion in a highly nervous individual. This conception is essential to a full understanding of the treatment of pain later described.

The control and relief of abdominal pain presupposes not only an accurate diagnosis, but a full appreciation of the structural and functional changes involved.

Since the pain is so important as a diagnostic sign, balanced judgment must at all times be exhibited whenever medication is to be instituted. No treatment for relief of pain must ever be permitted until the diagnosis has been made or at least until it seems reasonably certain that either the further study of the pain symptoms is unimportant or that the emergency demands immediate relief. This particularly pertains to the acute abdomen. The treatment of any symptom is, perforce, secondary to the contemplated treatment as a whole, nevertheless, pain as such is so important to the patient that definite measures must be instituted for its control.

Severe colic or pain of sudden onset is best relieved by  $\frac{1}{4}$  grain of morphine given hypodermically. If this does not alleviate the pain, it should be repeated within a half hour. In acute pancreatitis or ruptured peptic ulcer, there is evidence of a definite emergency and since surgery is necessary, scopolamin  $\frac{1}{450}$  grain may be added as it not only aids in relieving the mental anxiety, but prepares the patient for the subsequent anesthesia. If a gallstone colic or renal colic is diagnosed and no vomiting is present,  $\frac{1}{4}$  grain of morphine sulphate or  $\frac{1}{2}$  grain of codeine phosphate may be given by mouth. This is perhaps only justified if the patient is unduly apprehensive about a hypodermic.

Pain from appendicitis seldom requires a sedative and if it is severe enough, should then only be relieved when operation has been definitely decided upon. Pain due to torsion of

an ovarian cyst or fibroid on its pedicle or due to ruptured ectopic pregnancy requires morphine as early as feasible

In general, when a diagnosis of an abdominal accident is made surgery, when not contraindicated, should be instituted as soon as possible. When that decision has been arrived at morphine, pantopon or codein should be used to relieve the patient of all the suffering possible. One need not fear any harmful effect on the subsequent anesthesia.

In general or local peritonitis, where for some reason immediate operation is not deemed advisable, morphine should be used freely. Since the pain is frequently the sign of intestinal activity, it may remain as a guide of therapy. Peristalsis should be kept at a minimum of activity. In local peritonitis, as found in diverticulitis, milder sedatives may be used, especially when the sigmoid is involved.

The postoperative "gas" pains should, I believe, be treated more conservatively than is the usual practice. This scarcely requires consideration within the scope of this paper, but perhaps these few comments are in order. It is true that at times such vigorous enemas as milk and molasses or the commonly used 1-2 3 enema may give good results by producing peristalsis and the subsequent expelling of gas. These pains are usually due to lack of peristalsis and distention of loops of intestine. This dysfunction disappears within a few days after the normal tone and function have returned. Continued injections of various irritating fluids can certainly result in prolonged gas pain disturbances or worse still an aftermath of colon irritation which the patient will carry for a long time. It has been my experience that a rectal tube, an opiate, pituitrin, or prostigmin along with heat to the abdomen, when acceptable, will do as much or more good than laxatives and enemas and be followed by less intestinal invalidism later.

The management of the more chronic and recurrent abdominal distresses and pains requires the consideration of several therapeutic measures, namely, drugs for sedative and antispasmodic effect, diet, physiotherapy, and psychotherapy. These four measures are involved in varying degrees in the

proper and successful handling of peptic ulcer, irritable colon, chronic constipation, ptosis of the kidney and the other disorders mentioned

In simple uncomplicated peptic ulcer pain is almost invariably relieved by adequate alkali and food. By adequate alkali is meant sufficient amount to neutralize completely the acid gastric juice. A powder containing sodium bicarbonate 30 grains and calcium carbonate 30 grains taken in  $\frac{1}{2}$  glass of water will produce relief within five to ten minutes. One glass of milk and a few crackers will produce the same results. However, if the ulcer is in a fairly active state, the pain will recur after a time or usually again after a later meal and, therefore, the powder must be repeated. This procedure will not be effective as a management nor should it be continued as such since the patient will not be cured, but will be lulled into a state of false security. The control of the pain of peptic ulcer will be incorporated in the general management. In a properly managed ulcer diet the pain usually disappears within a day and seldom is it noted after the third day. In fact, when present after six to eight days, some complication should be suspected and searched for. When night pain is noted, alkalis should be given every hour or two throughout the night. This pain indicates obstruction at the outlet with an associated continued secretion. This latter complication will best be controlled by emptying the stomach with a tube at 10 P. M. and 1 or 2 A. M. Frequently pain that persists at night will be promptly relieved by instituting this procedure for three or four successive nights. Coarse and irritating foods frequently precipitate trouble and, therefore, should be avoided.

Other drugs than alkalis are seldom needed for control of pain. Occasionally tincture of belladonna in 10-minim doses three times daily is used. More often a sedative of 10 grains of sodium bromide given three times daily or a barbiturate will help control the apprehensive patient. Papaverin hydrochloride  $\frac{1}{4}$  grain, elixir phenobarbital  $\frac{1}{4}$  drachm taken three to four times daily will be effective.

I have never been convinced that atropine in safe doses could be used long enough to demonstrate any great clinical benefit

In the forme fruste type of perforation, where operation is not considered, the above-mentioned medications are used and in addition hot wet packs to the abdomen are effective. Aside from alkali no part of the management of ulcer is so essential and withal as poorly appreciated as rest and relaxation. If this could be definitely made a part of the management of all early ulcers, less ulcer invalids would develop.

The pain and distress of an irritable colon are due to a disorganized functioning of that organ. Varying states of tension and spasm in the musculature are present with resulting colic, pain, soreness and unrest. These subjective symptoms are best managed by such measures as will allay peristaltic unrest and tend to develop an orderly functioning of the colon.

For the acutely persistent pain or spasmodic colic, the patient is placed at absolute rest and heat is applied to the abdomen in the form of hot wet packs, hot water bags or electric pads. Tincture of opium 5 minims or camphorated tincture of opium 30 minims are given especially if there is an associated diarrhea. Codein or morphine are seldom necessary and then not to be given without the greatest certainty of diagnosis. Cathartics should never be given. The diet should consist of low cellulose residue as warm or boiled milk, soft eggs, toast and cooked cereals. As the colon becomes quiet, cooked fruits and cooked vegetables (all strained) may be given in increasing amounts until a normal diet is attained. This may require days or weeks of diet restriction and of careful food selection. Ice cold drinks, excessive fruit juices, bran, beer and buttermilk and specific foods to which the patient knows he is sensitive should be avoided. In the less severe pain the same dietary scheme should be used. Tincture of belladonna 10 minims and sodium bromide 10 grains, each three times daily after meals, will usually quiet the pains of flatulence and peristaltic activity. At times atropine  $\frac{1}{160}$

to  $\frac{1}{15}$  grain repeated four times daily will be more effective. If continued over any great length of time it may be found necessary to increase the dose. Syntropan has recently been advocated as an excellent antispasmodic.

The management of the type designated as mucous colitis is for all practical purposes the same. It seems scarcely ever necessary to resort to colonic irrigations or large water flushings either for treatment or the control of symptoms of either the ulcerative, noninflammatory or functional disorders of the colon. Should impactions develop due to improper management, enemas will be necessary for temporary relief, but this complicating trouble is obviated by the use of 2 to 3 ounces of warm olive or cottonseed oil as a retention enema when the bowel fails to move or the movement is hard and dry.

No specific dietary measures are to be followed for relief of pain as such in the treatment of disorders of the gallbladder, kidney or appendix or the several other conditions mentioned before.

Physiotherapy is considered frequently in management of the various chronic disorders of the abdomen not only as to treatment, but especially in the control of pain. The management of pain under consideration seldom requires infra-red or ultraviolet radiation and I have been so regularly disappointed in diathermy as a therapeutic agent in disorders of the colon as to abandon its consideration entirely.

Ice-bags to the abdomen in acute appendicitis remain in favor largely because presumably it is thought to limit the progress of the pathological process and also since it does seem to relieve pain. Heat to the abdomen will relieve pain in this disease, but is not to be recommended. Cold packs do not seem to be effective in any of the other disorders here considered.

Heat in the form of hot wet packs, hot baths, or electric pads comes nearer being the most specific physiotherapeutic measure available.

Hot wet packs should be applied continuously in local peritonitis of pelvic origin or in acute diverticulitis. Pain is

often readily controlled. They should be used in perigastrosis and where spasm and pain of ulcer are difficult to control. A hot tub bath of five to ten minutes' duration may be resorted to in the presence of kidney or gallbladder colic or severe spasm of the colon. The hot-water bag or electric pad is regularly used for the acute colics, but especially in the more chronic recurrent pains of the colon. In the latter condition heat is applied one half to one hour with a discontinuance for a like period of time, but to be continued for a longer time than the duration of the distress. Heat to the abdomen in the chronic intestinal invalid offers to that patient the greatest solace.

Massage is seldom to be recommended, although occasionally very light stroking over the colon by the experienced hand seems to be effective. Certainly deep, vigorous massage will aggravate rather than lessen the pain of colon origin. It should not be recommended in any other condition. I have suspected that the good noted by the patient has been largely psychic.

Pain from ptosis of the kidney or, as occasionally occurs, from ptosis of the stomach with an associated duodenal angulation will be relieved often by the wearing of a properly fitted supportive belt or corset. Special pads, cushions and straps seem to lend little more to the effectiveness of the corset than can be expected to come from the general support and relief from the tension on the attachments of the various organs.

The successful physician succeeds best who practices, knowingly or unknowingly, some measure of psychotherapy. The soothing influence from the comforting physician is never to be forgotten as one of the most effective therapeutic measures and pain in most instances is markedly increased by the fears of uncertainty. If the origin of pain can be definitely ascertained and proper measures of control instituted, a great part of the battle is won.



## CLINIC OF DR. J P GREENHILL

### COOK COUNTY HOSPITAL

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#### RELIEF OF PAIN ARISING IN THE FEMALE PELVIS

PATIENTS are more grateful for the relief from severe pain than for anything else which physicians can do for them. Fortunately in most instances, the cause of excruciating pain can be determined and removed. Thus, for example, the severe pain due to appendicitis, ruptured ectopic pregnancy, ruptured hollow viscus, etc., may be relieved by surgical operations. In other instances, excruciating pain can be relieved by the opiates as in cases of gallbladder colic, renal colic, etc. However, there are many instances where the source of annoying pain cannot be found or, if determined, it cannot be removed or satisfactorily relieved.

In the female pelvis the genital organs are a frequent source of severe pain. In inflammatory diseases of the tubes and ovaries, especially those due to gonorrhea, the pain may usually be relieved by conservative treatment. However, in many instances an operation is necessary to bring about relief. Following such operations, most of the pain disappears. Less frequent causes of pain in the pelvis are fibroids which produce pain by pressure on other organs or submucous fibroids which the uterus attempts to expel.

Endometriosis is another pelvic condition which usually produces pain. The pain associated with this abnormality as well as that due to fibroids may be relieved by surgical measures and sometimes by radiation therapy. Ovarian tumors occasionally cause pain and this pain may also be eliminated by removal of the growths.

There are, however, types of pain in the pelvis which are difficult to relieve. These are the severe dysmenorrheas, the



intractable pain associated with carcinoma of the cervix and other pelvic organs, and pain which occasionally follows operations in the pelvis and lower abdomen

**Dysmenorrhea**—Dysmenorrhea is generally divided into two types, primary and secondary. The latter is caused by pathologic conditions in the pelvis, usually uterine fibroids, endometriosis, or salpingitis. Surgical correction of the pathologic disturbance usually cures the menstrual pains. Primary dysmenorrhea, on the other hand, is not associated with any abnormality in the pelvis and its treatment is one of the most baffling problems with which physicians have to deal. Almost every type of analgesic drug has been employed to relieve primary dysmenorrhea, but not one has given any semblance of uniform success. There is no unity of opinion concerning the exact cause of this type of pain. Novak and Reynolds are of the opinion that the immediate cause of dysmenorrhea is an exaggerated contractility of the uterus, manifested by pain if the pain threshold is lowered or if there is an actual imbalance between the two hormones that appear to regulate this. These two hormones are the follicular factor, the normal stimulant of uterine excitability and progesterin, the normal inhibitor. Hence, Novak says in cases of dysmenorrhea it is advisable to administer biologic uterine antispasmodics and the one recommended is the luteinizing principle obtained from the urine of pregnant women. Other individuals believe that the strong pains associated with menstruation are due to an imbalance in the pelvic sympathetic system. Perhaps both factors are involved.

Because the exact etiology of dysmenorrhea is still unsettled, treatment is in most cases empiric and not satisfactory. There are a large number of young women who suffer inexorably every month in spite of what is prescribed. For those who are not helped by medication, physiotherapy, psychotherapy, dilatation and curettement and other measures, resection of the superior hypogastric plexus yields excellent results because in the large majority of cases instant relief is obtained. This operation will be described shortly.

**Pain Due to Pelvic Malignancy**—Carcinoma of the uterine cervix is one of the most serious afflictions a woman can develop because at least three out of every four women who have the disease die from it. Furthermore, nearly all of these women suffer excruciating pain during the latter part of their lives and in a large proportion of cases the pain is constantly present both day and night and is almost unbearable. This is due to the fact that the sensory nerves become involved in the malignant growth. There are at present three means of relieving this pain. The first and the one almost exclusively used at present is the administration of derivatives of opium, chiefly morphine. However, there are disadvantages to this form of therapy, particularly the necessity of giving constantly increasing doses as the patient's tolerance increases, the nausea and vomiting which some women experience, the idiosyncrasy of others, the addiction which many women develop, the excitement produced in some and the expense for poor patients. The second method of giving relief from pain is surgical and consists essentially of pelvic sympathectomy and chordotomy. The third means of relieving pain consists of blocking the nerves which conduct pain sensation. This may be accomplished by a number of different solutions, but alcohol is the most commonly used.

**Intraspinal Alcohol Injections**—The simplest of the aforementioned methods is the intraspinal (subarachnoid) injection of absolute or 95 per cent alcohol. It is based upon the idea of destroying some of the nerve fibers in the posterior nerve roots, these being the roots which convey the sensation of pain. Injection of absolute or 95 per cent alcohol into the subarachnoid space will prevent all painful peripheral stimuli from reaching the medullary centers, even if the stimuli act at the level of the spinal ganglia, the intervertebral foramina or the spinal roots. The technic of the injection is as follows. No preliminary medication is given because we wish to observe the immediate effects of the injection. Most patients with advanced carcinoma of the cervix and other genital organs have much more pain on one side than on the other. The

patient is placed on the side opposite to that where most of the pain is present. A pillow or pad is placed under the pelvis and side to elevate the sacral and lumbar portions of the spine, her back is arched as much as possible, her body turned somewhat ventrally and the head lowered slightly. By placing the patient in this attitude we raise the sacrolumbar region of the spine to the highest level and at the same time make the posterior or sensory nerve roots lie horizontally. The anterior or motor nerve roots come to lie in a plane which is usually out of reach of the alcohol. Even if the motor nerves are not removed from the field of the alcohol, they are not often affected because sensory nerves are more susceptible than motor fibers to the effects of alcohol.

Someone should hold the patient in the proper position. A weak solution of iodine or other antiseptic is applied over the lumbar and upper sacral regions. In most of my cases the fourth lumbar interspace is selected for the injection of alcohol and the results have been highly satisfactory. An ordinary lumbar puncture needle with a stylet is used. The needle is injected into the desired interspace just as for an ordinary lumbar puncture and I prefer not to use novocain in the skin before inserting the needle. After the needle is in the subarachnoid space, as evidenced by the flow of spinal fluid, 0.5 cc of absolute or 95 per cent alcohol is injected into the cerebrospinal fluid. For this purpose it is best to use a tuberculin syringe so as to be sure not more than 0.5 cc is injected. Furthermore, the alcohol must be injected very slowly, drop by drop, taking about two minutes for the injection of the 0.5 cc. This will avoid a mixture of the alcohol with the spinal fluid. The alcohol rises immediately to surround the posterior roots because the specific gravity of alcohol is about 0.806, whereas, that of the spinal fluid is 1.007. No attempt should be made to draw spinal fluid into the syringe to mix it with the alcohol because this is exactly what is *not* wanted. After the injection is made the needle is withdrawn and the puncture hole covered with sterile gauze and adhesive. Before the injection is completed, the patient will complain that the

upper leg feels numb or hot and that she cannot move the leg. The numbness is almost routinely experienced after the injection, but disappears spontaneously after a few hours or few days in most of the cases. In spite of what the patient says concerning her inability to move the leg, she can easily move it when requested to do so. At the same time that the patient informs us of the numbness she also often tells us either voluntarily or in answer to our query that her pain has disappeared. The longer the patient is permitted to lie on her side, the better the results. Hence I now keep my patients on their side for two hours after the injection. Then these women are permitted to get up and walk around. Some find difficulty in getting up from a chair because their "leg is asleep." Sometimes the leg feels heavy and the patient experiences some trouble in walking up steps because the knee flexes readily. These sensations usually wear off in a few hours, although in some women they last a number of weeks. Nearly all of my patients went home within three hours after the injection and no ill effects have been observed from this procedure. It is perhaps best, however, to keep the patient in a hospital for at least twenty four hours.

If the patient has pain on both sides, an injection is made a week later with the patient lying on the opposite side. The same amount of alcohol is injected.

Formerly I restricted the subarachnoid injection of alcohol to patients with hopelessly advanced cancer. Until we knew more about the effects of absolute and 95 per cent alcohol on the spinal cord I did not extend the use of this procedure to other cases. Recently we have obtained striking results in cases of pruritus vulvae.

Thus far my associate, Dr. Herbert E. Schmitz, and I have performed alcohol injections on 45 women who have advanced cancer of the cervix. We have completely or greatly relieved all but four of these patients. Some of these women have been free from pain for as long as ten months. A few who had pain on both sides and who experienced almost immediate relief on

one side after an injection, asked that the injection be repeated on the other side

**Pelvic Sympathectomy**—Another procedure which has yielded excellent results in cases of hopelessly advanced cancer of the genitalia is the operation known as pelvic sympathectomy, resection of the presacral nerve or resection of the superior hypogastric plexus. This operation may be performed by anyone trained to do abdominal surgery. The risk involved is slight, the technic is not complicated and local anesthesia may be used for most if not all of the operation, if desired.

The portion of the sympathetic nervous system which is removed is that known as the presacral nerve or superior hypogastric plexus. This plexus can readily be found if one looks for a triangle the base of which corresponds to a line uniting the two common iliac arteries at the level of the sacral promontory, the sides being these arteries and the apex of the triangle being the point of bifurcation of the aorta. This triangle occupies the lower third of the fourth lumbar vertebra, the last intervertebral cartilaginous disk and the fifth lumbar vertebra. The base of the triangle is about 7 cm in length and the distance from the base to the apex is almost 6 cm. A large part of the left side of the triangle is occupied by the left common iliac vein, which arises from the inferior vena cava and passes downward from beneath the right common iliac artery to accompany the left common iliac artery. The triangle is divided vertically into two equal halves by the middle sacral artery, which arises from the back part of the aorta just at its bifurcation and courses straight down to the upper part of the coccyx. Since this vessel is easily felt through the peritoneum it is often mistaken for the presacral nerve, which runs parallel to it. From the origin of the inferior mesenteric artery down to the bifurcation of the aorta, the sympathetic nerve fibers lie on the aorta, separated from it only by a layer of thin connective tissue. The branches of the plexus which accompany the left common iliac vein are separated from this vessel by areolar tissue, making elevation of the nerve easy. However, as the nerve fibers go still far-

ther down, they lie on the perichondrium of the last lumbar vertebra, and the cartilaginous disk between this vertebra and the sacrum. At this point the plexus lies above the middle sacral artery and veins.

The entire triangle is covered with peritoneum, hence the nerve plexus lies between the peritoneum and the underlying bones. The fibers are not adherent to the peritoneum, but are separated from it by more or less fatty tissue, depending on the obesity of the patient.

Two questions may properly be raised. One is whether the large fibers of the sympathetic system are really sensory. Ranson says that proof of this was offered more than twenty five years ago by Edgeworth and that he confirmed Edgeworth's observations. If the roots of the spinal nerves are cut proximal to the spinal ganglions, all the motor fibers degenerate, but the sensory fibers remain.

The hypogastric plexus produces vasoconstriction of the blood vessels of the internal genital organs and it inhibits the secretion of the genital glands, whereas the parasympathetic nerves produce the opposite effect. Section of the superior hypogastric plexus does not alter the normal menstrual cycle nor does it interfere with uterine contractions during labor. Likewise, section does not produce glandular atrophy or any disturbances in the motor function of the bladder or rectum. Therefore, the nerve fibers of the superior hypogastric plexus are sensory and not motor. They carry the sensations from the internal genital organs to the medullary centers. Hence resection of the portion of the superior hypogastric plexus above the hypogastric ganglion is a simple way of relieving a patient of severe pain arising in the pelvic organs.

The second question that may be raised concerns the possible harm done by removing a portion of the sympathetic nervous system. Ranson states that Cannon and his students completely removed from cats "the sympathetic chain on both sides, from the highest cervical to the lowest sacral ganglion. Such completely sympathectomized cats have lived under laboratory conditions for many months. Everything indicates that

almost any part of the sympathetic system can be removed without seriously endangering life”

**Technic of Pelvic Sympathectomy** —The technic of pelvic sympathectomy is as follows. The patient should be placed in the Trendelenburg position after a midline incision has been made from the umbilicus downward toward the pubis for about 10 to 12 cm. After the peritoneal cavity is opened, the small intestine is packed off and the sigmoid and rectum are pushed to the left side and held there with a wide retractor. The uterus, adnexa and bladder may then readily be inspected and palpated. One may detect a complication, that can be remedied by a surgical procedure. The region of the lower two lumbar vertebrae and the upper part of the sacrum is exposed to view. In thin women, it is possible in some cases to see the presacral nerve immediately beneath the peritoneum. Whether or not the nerve is seen, the parietal peritoneum above and in the middle of the sacral promontory is elevated and incised with scissors. This incision is extended upward for about 4 or 5 cm. and for a similar distance down along the sacrum. When the peritoneal flaps are pulled aside, a fibrocellular connective tissue layer will be exposed, covered by more or less adipose tissue. This tissue can easily be separated from the peritoneum and the lower end of the aorta without danger. It is in this layer that the presacral nerve lies. With an aneurysm needle the tissue is elevated at the bifurcation of the aorta and the dissection is carried to a still higher level. As this is done, it will be found that in most instances the tissue spreads out triangularly. The middle sacral artery should be pushed away from the nerve, but if it is injured it can readily be ligated.

After the dissection is carried as high as it is desirable to go, the layer of nerve tissue is separated from the underlying tissue down past the sacral promontory into the pelvic cavity. In this region the plexus has divided into two hypogastric nerves, hence it is necessary to dissect one of these nerves at a time. At least 2 or 3 cm. of each hypogastric nerve should be resected in addition to 4 or more centimeters

of the superior hypogastric and the intermesenteric plexuses. The fibrous tissue layer, which contains the hypogastric nerves, is much more resistant than that which contains the presacral nerve. As the dissection is carried out, nerve filaments projecting outward will be encountered. These should be followed as far laterally as possible before they are cut. In most instances, ganglions will be included in the resection. The dissected tissue should preferably be removed in one piece. It is not necessary or advisable to ligate the presacral nerve or the hypogastric nerves before cutting them, because the only blood vessels in intimate contact with them are insignificant vasa nervorum. Very rarely does one encounter bleeding that requires more than simple temporary pressure to check it. (When the mesosigmoid is very short, care must be exercised to avoid injury to the inferior mesenteric vessels.) After the nerve is dissected, the posterior parietal peritoneum is sutured with plain catgut and the abdominal wall is closed in the customary way.

Thus far my associate and I have performed 50 sympathectomies. We have found that if all women who have excruciating pain associated with carcinoma of the cervix are subjected to this operation only about 50 per cent will be relieved of their pain. However, if the patients are properly selected almost all will be freed of their pain.

The women who can definitely be relieved of their suffering by sympathectomy are those who have pain in the middle of the lower abdomen, pain low in the back, rectal tenesmus, bladder pain and pain associated with vesicovaginal fistulas. The women who cannot be helped much by sympathectomy are those who have pain in the sacrum due to fixation of the parametrium, pain referred from the region of the sacro-iliac joint into the thigh posteriorly and laterally, pain down the anterior surface of the thigh due to involvement of a gland in the obturator canal and pain due to hydronephrosis and hydro-ureter.

Pelvic sympathectomy is also curative of most cases of intractable dysmenorrhea where every form of therapy has



been tried and failed Naturally this form of treatment for menstrual pain is heroic and hence it should be used only as a last resort

**Chordotomy**—The operation known as chordotomy should be attempted only by one skilled in neurologic surgery The operation consists in the removal of a number of laminae, opening the dura mater and incising one or both anterolateral columns of the spinal cord as necessary If the incision in the cord is not accurately placed, the pain may not be completely relieved because insufficient fibers were sectioned or the motor pathways (pyramidal tracts) may be damaged, resulting in paralysis of the legs and interference with sphincter control I have had no personal experience with this operation

**Pain Following Operation**—There are occasional instances where distressing pain either arises or persists following an operation on the pelvic organs or in the lower abdomen If a second operation is performed because of the pain it seldom gives relief because usually no cause for the pain can be found In most of these patients, relief from the pain can be obtained by a pelvic sympathectomy

**Conclusion**—Any procedure which will relieve women of excruciating or constant pain is worthy of a trial For the severe pain which is associated with malignancy of the female genitalia, drugs are unsatisfactory There are three operations which can give most of these women relief In the order of their simplicity they are the intraspinal (subarachnoid) injection of absolute alcohol, pelvic sympathectomy and chordotomy The subarachnoid injection of alcohol should be tried first because it entails the least risk and gives relief in the vast majority of cases Resection of the superior hypogastric plexus is more risky because it necessitates opening the abdominal cavity, but the risk is slight and the operation can be performed by anyone with surgical experience Chordotomy on the other hand requires special technical knowledge

In addition to the unfortunates who have advanced carcinoma there is a group of women who suffer inexorably during each monthly flow For those who have not been helped by

medication, dilatation and curettement and other means, the operation of pelvic sympathectomy offers instant relief in the majority of cases. Likewise this operation will prove helpful in the cases where annoying pain follows an operation in the pelvis.

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## CLINIC OF DR ISADORE PILOT

### COOK COUNTY HOSPITAL

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#### SEPTIC SORE THROAT CLINICAL AND BACTERIOLOGICAL CONSIDERATIONS

A SEVERE type of tonsillitis is usually designated as septic sore throat. Such marked reactions in the throat are particularly striking in infections due to the hemolytic streptococci. From the point of view of serious and fatal complications the significant septic sore throat has been observed in epidemic form, as milk-borne septic sore throat. Most sporadic cases appear as a mild sore throat or tonsillitis, but occasionally a similar intense type is encountered resembling in severity the epidemic form but not related to milk. The complications in such sporadic cases are often serious and deserve our special consideration.<sup>1</sup>

Acute throat infections present an extremely variable clinical picture. Several factors determine the local and general symptoms. These are, the virulence of the organisms, the dosage of these bacteria, the amount of lymphoid tissue in the oropharynx and most important the reactivity of the patient. Variations in these factors result in either mild or severe sore throat. A large dose of virulent streptococci in a patient with considerable lymphoid tissue and a lowered general resistance lead to a marked sore throat and general symptoms of sepsis that are characteristic of septic sore throat.

**Etiology**—The bacteria most often involved in throat infections are the hemolytic streptococci. In our experience 50 per cent or more of the sore throat encountered in daily practice appear to be due to the streptococci. Apparently all human types of *S. hemolyticus* are responsible. The strep-

tococci yielding scarlet fever toxin cause sore throat with a characteristic rash of scarlatina in susceptible persons. In the Dick negative the streptococci may still be responsible for sore throat of a variable severity. Severe septic types are often associated with the scarlet rash and in their tendency to complications and sequelae septic sore throat and severe scarlet fever behave alike. The streptococci of erysipelas are also capable of producing sore throat. These strains are more difficult to identify. Erysipelas may originate from these streptococci invading the skin possibly through an abrasion about the nose or ear, particularly if there is a sinusitis or purulent otitis media that complicates the throat infection.

The most severe type is observed in acute throat infections due to the encapsulated mucoid hemolytic streptococci known as *Streptococcus epidemicus* (Davis). These peculiar properties identify these streptococci readily and have aided us greatly in investigating sporadic sore throat. In milk-borne epidemics these streptococci have always been the source of the mastitis in the cow whose udder sheds millions of these bacteria per cubic centimeter of milk. When raw milk containing *S. epidemicus* is consumed the dosage of infection is massive and a very severe type of sore throat infection results. Serious complications and an occasional death are associated with most milk-borne epidemics. In sporadic cases due to *S. epidemicus* the mode of infection is not through milk but through carriers or active cases. The dosage of the infecting bacteria is much smaller therefore, than in the epidemic type and the reaction of the throat milder. Occasionally the sore throat in the sporadic case may be severe and dangerous. Indeed when a very severe sore throat is observed, the encapsulated streptococci should be suspected. In a series of streptococcic sore throats of the sporadic type 10 per cent of the strains proved to be *S. epidemicus*. The complications and sequelae in this smaller group were more numerous and serious than in the other 90 per cent.<sup>2</sup>

A large number of the streptococci causing sore throat are not identified with *S. epidemicus*, *S. erysipelatus* or *S. scar-*

latinae. Little is known about their epidemiology and distribution. They may have considerable virulence and may cause sore throat and complications like that of the specific streptococci.

Among the organisms other than streptococci causing sore throat the pneumococci are most serious and often not recognized. These encapsulated diplococci are often very virulent and responsible for an infrequent but severe sore throat that may be very septic in its behavior. In two instances the pneumococci were recovered from the throat and the suppurative lesions of the neck in patients who were desperately ill. One succumbed and at autopsy the pneumococci were found in the pus associated with an extensive thrombophlebitis of the common facial vein with its tributaries, and in the embolic abscesses of the lungs. *Staphylococcus aureus* was isolated in pure form in four cases of sore throat.<sup>2</sup> Together with the nonhemolytic streptococci they are the cause of a mild form of sore throat. The diphtheria bacilli and Vincent's spirochetes are responsible for a characteristic variety of sore throat, but often the associated streptococci and pneumococci may modify the throat condition and render the condition more septic.

The essential changes in septic sore throat occur in the lymphoid ring of the pharynx. The faucial tonsils become enlarged, edematous and purulent exudation develops on the surface and in the crypts. If the faucial tonsils have been previously removed the lymphoid tissues of the pharynx and at the base the tongue become similarly involved. The spread of infection may be in one of several directions. The extension through the nasopharynx, the eustachian tube, middle ear, mastoid and meninges has been well recognized and emphasized. The pathogenesis of general sepsis has been less clearly understood. So-called "primary streptococcus peritonitis" has been ascribed to a primary hematogenous infection from the throat. Recent studies have pointed out the possibility of the streptococci passing through the depressed gastric juice unharmed to the intestine where they migrate

through the wall to the peritoneum. The spread to the neck and large veins has also been recently pointed out. Fatal cases of septic sore throat have been assumed to be the result of a septicemia from the throat, but careful study has shown that many develop sepsis from a thrombophlebitis that extends from the veins of the tonsils to the common facial and jugular vessels. The streptococcic pneumonia may therefore be embolic in origin, as well as the result of aspiration or lymphatic extension along the bronchi.

**Symptoms**—It is needless to describe the symptoms of ordinary sore throat, but the striking features of the more severe and septic cases should be pointed out. At the onset the fever may be high and pulse very rapid. In one instance the temperature was 107° F. Upon examination early the only finding in the entire examination may be a redness of the lymphoid structures of the mouth. Swelling from edema and purulent exudation develop in twelve to twenty-four hours. White patches and intense redness are noted on the second and third days. The membrane in a streptococcus throat as a rule is readily wiped away although the orifices of the crypts may still appear as white spots after swabbing. The pneumococcus produces a more adherent membrane resembling in this respect diphtheria. Fever and tachycardia are sustained for three to five days and then drop slowly in two or three days. Within this period adenitis, otitis media, peritonsillar abscess, parapharyngeal abscess, cellulitis of the neck, or peritonitis may develop. Complications should be suspected if the patient is unable to open the jaws freely and tenderness and induration appear at the angle of the inferior maxilla. In some instances with suppurative processes in the loose tissue of the neck, the lower jaw may be so restricted in its motion as to render examination of the throat difficult. With complications, the fever and pulse rate remain high and the septic state more marked. A severe chill or chills may be indicative of thrombophlebitic involvement of the veins of the neck. In such instances pains in the chest, signs of

pulmonary involvement may appear quickly and lead to a fatal termination

In patients without tonsils the clinical picture due to identical bacteria is modified. No doubt tonsillectomized persons have less sore throat, but instead many have "colds" and upper respiratory infections. In these individuals the streptococcus infection manifests itself usually with an abrupt onset, chilliness, fever  $102^{\circ}$  to  $105^{\circ}$  F and rapid pulse. Prostration and muscular pains are marked as in influenza, but the pulse is rapid and not relatively slow in proportion to the temperature. Upon examination there may be no findings except for some redness of the pharynx. On the following day the lymphoid follicles may become swollen and covered with exudate. Soreness of throat as a symptom often is absent or only evident as a raw feeling at the base of the tongue. Cough and chest pains may develop, but the lungs and pleurae yield no signs of involvement. Pleuritis and pneumonia, in spite of these common symptoms, are unusual complications. Adenitis is less common than in patients with tonsils, but otitis media is not infrequent. In our experience cellulitis and thrombophlebitis of the fatal type are observed only in the tonsillar forms of septic sore throat. Persons with tonsillar tags develop sore throat with a clinical picture in relation to the amount of lymphoid tissue still present between the tonsillar pillars. Usually the tonsillar remnants become swollen, red, and covered with exudate.

**Complications**—These most often develop during the first week of illness. Occasionally after a period of convalescence of a week or two a recurrent sore throat, fever and complications of adenitis, otitis, etc., may arise. At this stage, as the result of a possible state of hypersensitiveness to the streptococci, polyarthrits, endocarditis resembling the rheumatic type may ensue. In a similar way, erythema nodosum and acute glomerulonephritis may appear as sequelae. In the patients with late complications and sequelae a careful examination of the flora of the throat will reveal the persistence of



the hemolytic streptococci in large numbers in swab cultures of the throat, but none in cultures made of the blood<sup>1</sup>

**Diagnosis**—The diagnosis of septic sore throat is not difficult. Bacteriological examination to exclude diphtheria is very important. To demonstrate the causative bacteria, particularly the hemolytic streptococci, swab cultures should be made of the exudate of the throat, avoiding contamination from the saliva and sputum. The swab should be streaked on the surface of blood-agar plates and in addition inoculated into melted tubes of blood agar which are poured into petri dishes. The latter method will yield hemolytic streptococci that may be overgrown by other organisms on the streaked plates. The poured plates also yield a more accurate picture of the relative number of streptococci. To bring out the mucoid encapsulated streptococci (*S. epidemicus*) ascites fluid is added to the infusion blood agar in proportion of 1 part of ascites fluid to 5 of agar. In severe cases with complications the *S. epidemicus* is a frequent offender because of the more invasive character of the encapsulated organisms. If sore throat appears in epidemic form, the epidemicus should be suspected and if found milk should be suspected as the source of the epidemic infection. No epidemics have been reported as yet from pasteurized or certified milk. Extensive epidemics continue to be traced to raw milk derived from cattle with mastitis due to *S. epidemicus*.

The diagnosis of streptococcus sore throat may be difficult in connection with tonsillectomized persons. Undoubtedly many person with respiratory infections are designated as influenza which in reality are due to the *Streptococcus hemolyticus*. If clinical features are inadequate for differentiation, a swab culture of the throat for hemolytic streptococci may aid in deciding the true clinical state.

In severe cases with apparent complications the recognition of peritonsillar abscess, thrombophlebitis of the vessels are exceedingly important. A chill, high fever, leukocytosis, inability to open jaws, pain and tenderness in the tissues about the angle of the jaw, all point to serious involvement. The

diagnosis of thrombophlebitis is not easy, but if definite should be made quickly and decisively so that surgical ligations and drainage may be immediately instituted

**Prophylaxis and Treatment**—In the epidemic form the source of septic sore throat is raw milk from a cow with mastitis due to a hemolytic streptococcus identified as *S. epidemicus*. To prevent further spread milk should be boiled until the responsible cow is located and excluded. In the sporadic form, sore throat is spread by contact with patients or carriers of dangerous streptococci. It is necessary, therefore, to isolate acutely affected patients exactly as in scarlet fever. If the patient is placed in the hospital because of the complications, these precautions should be insisted upon in order to protect other patients.

In active management of a patient the same measures are carried out as in ordinary tonsillitis. The patient should be watched closely for the development of complications. No specific antistreptococcus or antitoxic serums are available for all cases and in their absence one would be justified to employ a polyvalent streptococcus serum or even the scarlet fever antitoxin in the more serious cases, particularly when complications develop. In some instances it has been possible to employ convalescent serum or blood transfusion from persons who have recently recovered from sore throat. Our experience with convalescent scarlet fever serum in severe complications from septic sore throat is limited.

The treatment of the suppurative complications of the neck and the thrombophlebitis of the facial veins require the services of a surgeon who has thorough knowledge of the anatomical relationships of the structures of the neck to the lymphoid tissues of the throat. Ligation of the facial and jugular veins in severe septic types with chills may be a life-saving procedure.

Convalescence in sore throat is often neglected and may result in the appearance of complications and sequelae in the third and fourth weeks after the onset. If the throat remains deep red, the tonsils large and edematous, there is good indi-

cation that the streptococci are still present in the throat and capable of causing additional trouble. A swab culture should be made to ascertain their presence. If large numbers appear on the blood-agar plates, the patient should not be permitted to return to his activities and should be watched carefully. Often with the utmost precautions these carriers will develop polyarthrititis, endocarditis, erythema nodosum or acute glomerulonephritis. Antiseptic gargles should be tried in an effort to influence the carrier state. If the patient is tested with a killed suspension of the streptococci intradermally he may yield a strong reaction indicating a possible state of allergy to the streptococci. In such instances desensitization may be tried by administering minute doses of the vaccine intradermally or subcutaneously. Sensitization may be so marked that extremely small doses, 100,000 or less, are necessary to avoid reactions.

Following the acute and the allergic stages of sore throat, the throat may still harbor streptococci in the tonsils. Such throats may reveal enlarged red tonsils and may be associated with arthritic pains, and enlarged cervical glands. These persons are carriers of dangerous organisms that may spread the disease to others. After at least an interval of six weeks following the acute sore throat a tonsillectomy will terminate the carrier state and frequently relieve the patient of his joint pains.<sup>3</sup>

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## CLINIC OF DR SAMUEL J PEARLMAN

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### THE TREATMENT OF SEPSIS AND PYEMIA FOLLOWING TONSILLAR INFECTIONS

"IN hunting for pus deep in the neck find the Lincoln Highway—that is, the carotid sheath—and follow it. It is the natural highway for pus and for the surgeon in pursuit of pus.

"All of us have known almost from the cradle, that the great vessels of the neck are found under the inner edge of the sternomastoid muscle. It took me a long time, however, to sense the fact that the posterior belly of the digastric muscle is an equally sure guide to the great vessels high in the neck."—Mosher

Sepsis may be defined as the presence of bacteria and their products within the circulation. By pyemia is meant the presence of bacteria in the blood stream and their localization in distant parts of the body with the formation there of abscesses. In the last decade and a half, it has become increasingly evident that sepsis and pyemia not rarely follow acute tonsillitis and acute suppurations about the tonsils. If recognized early and treated efficiently, the mortality from these severe systemic infections can be considerably lowered. Before considering the treatment which is always surgical, a brief review of the anatomy of the involved region, the pathology and the manner of spread of these infections is desirable.

The tonsil lies upon the superior constrictor muscle, separated from it by a layer of connective tissue which is part of the pharyngeal aponeurosis. In surgical removal of the tonsil, a portion of the aponeurosis, dense in character, clings to the tonsil and is called the "capsule."

When a tonsillar infection spreads beyond its original confines, that is, when a tonsillitis becomes a peritonsillar abscess, the new inflammation is confined to the connective tissue above described, between the tonsil and the superior constrictor muscle

Should one of these extratonsillar inflammations spread and in one way or another pass through the superior constrictor muscle, the process is now in the deep tissues of the neck in an area called the parapharyngeal or pharyngomaxillary space or fossa. It is the invasion of this territory which is characterized by signs of sepsis in addition to other symptoms and which is so dangerous to the life of the patient

The anatomy of the parapharyngeal space or fossa has been concisely and admirably described by Mosher. "The inner boundary of the fossa is the superior constrictor with the tonsil attached to it. The outer boundary *below* is the internal pterygoid muscle lining the inner surface of the ascending ramus of the jaw and mating the masseter on the outside. The outer boundary *above* is the parotid gland, the gland at this point not being covered with fasciae. Posteriorly, the prevertebral muscles and the prevertebral fascia bounds the fossa. The fossa is divided into unequal parts by the styloid process and the muscles rising from it. These are in front of the great vessels and protect them. On the posterior wall near the middle line on the front face of the second cervical vertebrae, there are two lymphatic glands which drain the nose and the upper pharynx and are in chain with the deep cervical glands."

The anterior or muscular or prestyloid compartment contains no large vessels or nerve trunks. It communicates with the parotid space through a defect in the fascia of the latter and is in close relationship to the submaxillary fossa.

The posterior, vascular or retrostyloid compartment contains the carotid arteries, the jugular vein, the glossopharyngeal, vagus, hypoglossal, spinal accessory and the cervical sympathetic nerves.

It is clear that pus in the posterior or vascular compart-

ment, hence, has a clear pathway from the skull and its contents above, to the mediastinum below

There are at least three ways by which a tonsillar or peritonsillar infection may invade the parapharyngeal space. The first is by means of a retrograde thrombophlebitis of the tonsillar veins to the internal jugular vein. The second is by

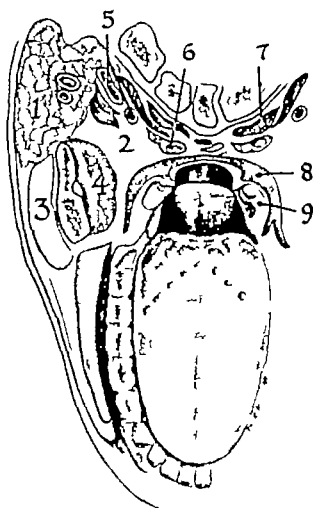


Fig 127.—Cross section of the neck at through the second cervical vertebra showing the pharyngomaxillary or parapharyngeal space. (After Mosher)  
 1 Parotid gland 2 parapharyngeal space 3 masseter muscle 4 Internal pterygoid muscle 5 great vessels 6 lymph nodes 7, prevertebral muscles 8 superior constrictor muscle 9 tonsil

way of a lymphangitis and lymphadenitis producing, because of the close attachment of the deep cervical glands to the internal jugular vein, first of all a phlebitis and then a thrombosis of the vein. The phlegmonous changes following a lymphangitis may also attack the veins secondarily. Finally, by direct extension, through the superior constrictor muscle, pus

from a peritonsillar abscess or pus produced by breaking down of inflamed glands, may involve the fossa and bathe its contents, attacking with ease the jugular vein because of its thin walls. Combinations of all these three types probably exist in most instances

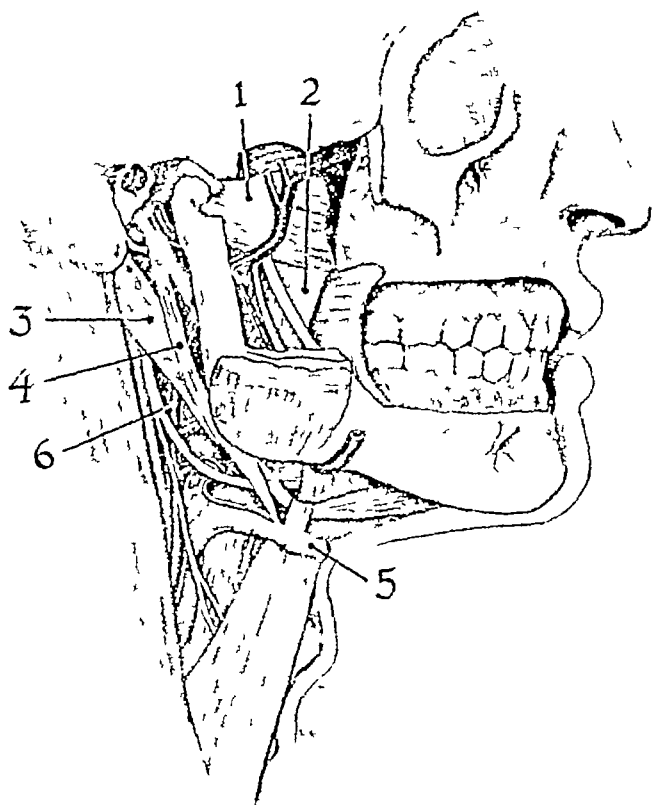


Fig 128—Dissection of lateral aspect of neck and pterygoid region (After Bock) 1, External pterygoid muscle, 2, internal pterygoid muscle, 3, digastric muscle, 4, stylohyoid muscle, 5, hyoid bone, 6, external carotid artery

The symptomatology of parapharyngeal space infections may be best handled, perhaps, by describing a typical case. Following an attack of tonsillitis, a patient develops a peritonsillar abscess. The usual treatment directed toward this condition fails to give speedy relief. Inability to open the mouth widely or trismus appears. Now trismus is a common

symptom of peritonsillar abscess, but it disappears quickly after drainage has been instituted. Its persistence, in spite of adequate drainage, or its reappearance after having previously disappeared frequently means that the infection has spread beyond the usual area involved in the ordinary quinsy. Its presence in parapharyngeal space infections is due to an inflammatory infiltration involving the internal pterygoid muscle which forms one of the walls of the space.

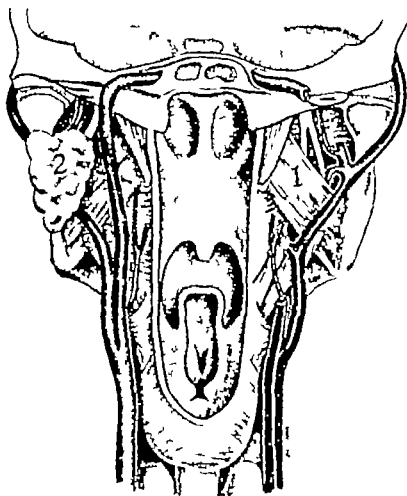


Fig. 129.—The parapharyngeal space from behind. The pharynx has been opened (After Bock.) 1 Internal pterygoid muscle one of the boundaries of the space, 2 parotid gland showing its close relationship to the space.

Fever soon appears. It is usually septic in type, but may be high and continuous. Chills are usually present and come with increasing frequency. A chill at the onset of a tonsillitis or peritonsillar abscess is not uncommon. Repeated recurrence of chills, however, is an ominous sign, and should cause the physician to extend every effort to arrive at a satis-



factory explanation He must be sure to rule out leukemia, agranulocytosis, pneumonia and all other conditions which could produce this state of affairs

Most of the time in addition to above symptoms, a brawny induration develops in the neck about the angle of the jaw. Rapidly progressing types and those in which the chief pathology is a retrograde thrombophlebitis from the tonsillar veins to the internal jugular vein may show little or no external swelling in the neck. They may, however, show some tenderness along the course of the vein. The more slowly progressive and benign forms, as a rule, are accompanied by the above described induration. Careful inspection of the pharynx, furthermore, may show some bulging of the lateral pharyngeal wall on the affected side, and the homolateral soft palate may be sluggish in its movements. In addition, there will be considerable pain and difficulty on swallowing.

Having established to his satisfaction that the deep tissues of the neck have been invaded, what is the surgeon to do? The treatment resolves itself chiefly into two parts. Pus is to be evacuated and a thrombosed jugular vein must be ligated or rather resected. In most uncomplicated instances, adequate drainage is sufficient to cause a disappearance of the symptoms. If, following drainage, there is not the expected relief, the internal jugular vein must be inspected and properly handled.

In those cases wherein the pathology is primarily a thrombosis of the internal jugular and there is no evidence of pus in the parapharyngeal space, one proceeds at once to the resection of the vein.

Some authorities insist that in addition to the above the tonsil on the diseased side should also be removed. In the event both tonsils are involved, it becomes more difficult in the absence of external signs to determine which internal jugular vein is involved. It is recommended under these circumstances first to explore the side on which the tonsil appears to be most involved.

Pus may be reached and evacuated in a number of ways

The abscess may be evacuated by way of the pharynx. After preliminary tonsillectomy a long curved hemostat is thrust at the point of greatest bulging through the superior constrictor muscle into the parapharyngeal space. Such a procedure, according to Hall, will in all probability reach only the anterior compartment of the space. A general anesthetic should not be used, for the danger of asphyxia is great. Infiltration anesthesia is risky because additional infection may be carried through the infected field into the deep tissues. It is better

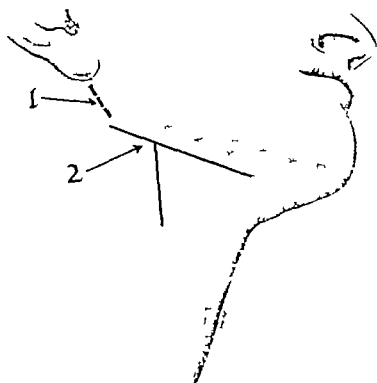


Fig 130.—1 Incision for "simple" incision. In the illustration the dotted line is somewhat long and too high. 2 Incision as advocated by Mosher.

to apply cocaine locally, and in addition to obtain some blunting of sensation by anesthetizing the sphenopalatine ganglion as recommended by Guttman for the opening of peritonsillar abscesses. It is obvious that the pharyngeal approach is of limited value and is not applicable in the absence of a pharyngeal swelling, it needs to be preceded often by a preliminary tonsillectomy, frequently in the presence of a peritonsillar abscess.

The external approaches are usually favored by men of

experience They should be used most of the time and when there is no pharyngeal lead or when the results of opening by way of the pharynx are not satisfactory after a wait of twenty-four to forty-eight hours The "simple" external approach may be used at times with satisfaction The angle of the mandible is found two fingerbreadths below the lobule of the ear and on a line with the mastoid tip A vertical incision is made just behind the angle through the skin and subcutaneous tissues A curved hemostat or scissors carried in an upward and inward direction through the deep fascia on to the internal aspect of mandible, *i. e.*, along the internal pterygoid muscle (Batson), enters the parapharyngeal space Neither of the two above approaches permits inspection or treatment directed toward the internal jugular vein

On the continent, especially in the Germanic countries, the approach popularized by Marschik is used almost exclusively The skin incision is along the anterior border of the sterno mastoid muscle, with its middle at the level of the angle of the mandible The deep cervical fascia is exposed, and at the level of the angle of the jaw is incised or opened by blunt dissection in the direction of the skin incision Further orientation depends on finding the stylohyoid muscle and the tendon of the digastric muscles These are beneath the angle and run from above and behind downward and forward At this point the tendon of the digastric perforates the fibers of the stylohyoid Both muscles are retracted downward Upward and forward blunt dissection exposes the anterior parapharyngeal space Blunt dissection with a forceps or finger beneath the digastric carries one to the posterior parapharyngeal space and the carotid sheath Following the stylohyoid the finger palpates the styloid process and may be carried upward to the base of the skull

In this country, Mosher's advocacy of the submaxillary fossa approach to pus deep in the neck has made a deep impression He recommends a generous T-shaped incision The skin flaps are retracted and the lower border of the submaxillary gland defined The gland lies between the two bellies of

the digastric muscle as in a sling, and is lifted upward. The facial vein is tied and cut. The finger or blunt forceps carried upward beneath the posterior belly of the digastric muscle finds the carotid sheath and pus if it is present. The styloid process is easily palpable and pus accumulations about the base of the skull may be reached. This exposure, better than the preceding one, allows for examination of the base of the tongue, and the floor of the mouth as well as the entire neck.

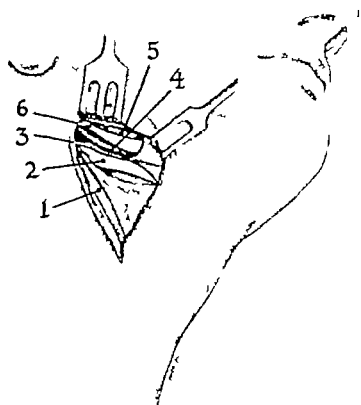


Fig 131.—Operative approach according to Marschlik. (After Wessely)  
 1 Sternomastoid muscle 2 digastric muscle 3 stylohyoid muscle 4 styloglossus muscle 5 stylopharyngeus muscle 6 loose fascia of the pharynx

from the base of the skull downward. The internal jugular vein is easily inspected and ligation and resection carried out (See Fig 130).

Finally, the parapharyngeal space may be exposed after Kramm's recommendation by a retromandibular approach. This has the claimed advantage of exposing the parapharyngeal space from the base of the skull to the angle of the mandible, and is said to be quite easy to do. The incision is car-

ried through the skin at the anterior border of the sternomastoid muscle from the tip of the mastoid to 2 cm below the angle of the jaw. Great care is necessary at this point to define the lower pole of the parotid gland, which is freed by blunt dissection and carried forward. Beneath and medial from the sternomastoid muscle the posterior belly of the digastric muscle now appears.

Somewhat deeper and more medial the internal jugular vein appears. One or 2 cm in front of and somewhat medial



Fig. 132.—Dissection of parotid region. (After Tandler.) In the exposure as advocated by Kramm, the lower and posterior border of the parotid gland is dissected free and pulled upward and forward.

to the vein the styloid process is opened by blunt dissection and the process cleared from its tip upward as high as possible.

A small or medium-sized nasal speculum is passed into the prepared cavity with blades closed. When the speculum is opened the styloid muscles separate, the styloglossus anteriorly, the stylohyoid posteriorly, and the whole anterior parapharyngeal space is exposed. Further use of the speculum exposes the vascular sheath back of the styloid process.

The advantage of this exposure is that it brings one to the

center of the infected area. The greatest difficulty is encountered at first in freeing the lower pole of the parotid gland. Thereafter the dissection proceeds with ease. The styloid process, a most important landmark in these exposures, is easily exposed and no large vessels are in the way excepting the internal jugular vein which one is anxious to see and handle surgically, if necessary.

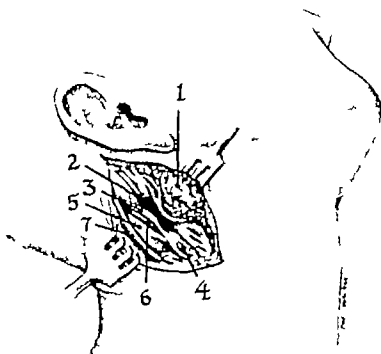


Fig 133—Exposure of parapharyngeal space as advocated by Kramm  
1 Parotid gland 2 parapharyngeal space 3 styloid process 4 stylohyoid muscle 5 digastric muscle 6 internal jugular vein 7, sternomastoid muscle

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## CLINIC OF DR. CLARENCE F G BROWN

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### JAUNDICE A BRIEF DISCUSSION OF DIAGNOSIS FOLLOWED BY A PROPOSED MEDICAL MANAGEMENT

THE principal decision which a physician should hasten to make when he sees a patient with jaundice is whether to advise surgery or try medical management. It is just as important in this year of progress and laboratory enlightenment to elicit a most detailed and careful history as it ever was before. The interpretation of the many tests to measure liver function still depends on the clinical conditions as shown by the history, the physical examination, roentgen studies, and the usual laboratory procedures. If surgical measures are finally chosen, it is usually our experience that patients seem instinctively to refuse or defer an operation in painless jaundice. This patient deferred several times. Medical management must be advised not only to help the condition if it can be helped, but to uncover latent symptoms so that the conditions difficult to diagnose, primary carcinoma of the bile tract, carcinoma of the head of the pancreas, and silent stone, will not be allowed to develop past the point of helpful surgery.

The patient I am showing you today illustrates the necessity of briefly reviewing the increasing physiologic knowledge of the liver and bile tracts so that we may benefit by better clinical judgment. Today we can bring out only an occasional point so completely discussed by Ivy<sup>1</sup> in his recent review with a bibliography of 553 articles covered. Each year, through advances in the fact finding sciences of physiology and pathology, we are able either to discard or justify our remaining clinical concepts.



The decision as to policy of treatment which we faced in this patient is a common dilemma. Even with our better understanding of liver processes it is still difficult to be correct in the choice of either medical or surgical treatment of jaundice. To illustrate these points in differential diagnosis I will tell the story of C S.

This strongly built, well-nourished man, fifty years of age, came to our clinic in February, 1933. He was deeply jaundiced, had no pain, not very sick, and complained only of some fatigue, belching and constipation. He remembered having a painful jaundice five years before which cleared up in a few weeks without surgical aid. As he recalled his sensations in the previous attack, he did not seem much impressed by the pain but always pointed to the right costal margin. He did not recall radiation to the shoulder blade. It was definite that the first attack under our observation was at least less painful as he senses only an epigastric fulness. He stated that he lost 40 pounds during seven weeks. In December, 1932 he lost his appetite. Following this, the jaundice began with a slow insidious onset. He was never seriously ill nor had he ever noted any gastro-intestinal symptoms except for his 1928 jaundice. He had been in perfect health from 1928 to December, 1932. There was nothing in his habits or occupation to suggest contact with carbon tetrachloride, chloroform, tribromethanol, phenylhydrazine, phosphorus, or trinitrotoluene. He was a machinist, never drank alcoholic beverages, not even coffee. He went to bed early, had a happy home life with good cooking and never worried. As he never took any kind of medicine we could rule out the other toxic jaundices due to the cinchophens and arsphenamines. He had eaten few carrots and no mushrooms. Some of the above conditions have been discussed by C A Elliott<sup>1</sup> and Walter Nadler,<sup>2</sup> Starr,<sup>3</sup> Bloch and Rosenberg<sup>4</sup> and Carroll,<sup>5</sup> which form a good background for clinical understanding of the liver and bile tracts.

Physical examination showed no fever or enlargement of the spleen. There was no history of intermittent fever with chills as with a "ball valve" stone in the common duct. He did not have the greenish-yellow color as in Gaucher's disease and the normal spleen definitely ruled that out. A negative Wassermann test turned our minds from a luetic cause of jaundice. A coincident Banti's disease was ruled out by the absence of spleen enlargement and hypochromic anemia. There were no other signs of decompensation to go with the jaundice associated with a failing heart. There were no enlarged collateral veins visible beneath the skin of the abdomen and not enough enlargement of the liver to suggest cirrhosis. The liver edge was smooth, enlarged three fingers and very faintly tender. There were no hard nodules and not enough enlargement of the liver to suggest the jaundice from a liver so full of metastases as to bring on jaundice. This condition is usually quite terminal and easy to diagnose by a careful search. The gallbladder could be palpated and was not tender. With no fever it is hard to conceive of yellow fever. I have seen a patient with undulant fever who had coincident mild jaundice but again we can rule this out.

The usual blood studies ruled out the jaundice with pernicious anemia sickle cell anemia and very probably hemolytic jaundice. The patient had never noted blood in the urine and our tests showed none so that paroxysmal hemoglobinuria could be ruled out. Malaria was not found and there were no symptoms or history suggestive of "blackwater fever." The liver did not seem large enough to consider this a typical example of Hanot's cirrhosis. Apparently obstructive back pressure must either be quite marked or of a long duration or occur in a vulnerable liver to produce this old clinical picture. In Weil's disease there is usually fever albuminuria and more malaise and aches than this patient had. After considering this array of clinical disorders together with a few not mentioned such as Laennec's cirrhosis and Hodgkin's disease I think you will agree in a movement toward pure pathology and physiology to simplify such a subject as this.

There are several diseases not yet ruled out even with the complete gastrointestinal roentgen study. These are aneurysms of the hepatic artery dormant echinococcus cyst and peritoneal adhesions. Primary tumors of the gall bladder or bile tracts or the pancreas are consistently missed by the best roentgenologists. Gallbladder visualizations are risky procedures in jaundice. One was done on this patient largely for academic purposes after the jaundice had subsided somewhat. No stones were seen the gallbladder partially filled and was very slow in emptying.

Surgery was advised because of a provisional diagnosis of possible carcinoma, although the history would indicate a silent stone with partial obstruction and secondary hepatitis. The patient decided to assume the risk of deferring the operation which gave us the opportunity of using the usual liver function tests. Time does not allow the interpretation of each one during the course of observation from February 1933 to May 1935. During each of the three attacks which occurred the urine was dark the stools light. The gastric contents were examined frequently for the two year period and showed a free acid of 40 to 60 at first gradually falling to 30 to 40 since August 1933. During the first three weeks after each attack the benzidine test was positive in the stomach contents and stools and at other times was negative.

Recently there has been an increasing interest in liver function tests accompanied by a widespread increase in their uses. These tests are certainly an effort in the right direction. Many times they are ordered by a busy physician as part of his ritual, in much the same way as useless metabolic rates. They may be blamed afterward for a bad decision which should have been based on more simple and obvious observations. As it turned out later in this instance they, together with the time elements, helped guide us to a better preoperative diagnosis than the provisional one of carcinoma. Some of them require a highly trained laboratory staff and some are so complex as to lead to error. The interpretation

is extremely confusing in a mixture of pathological conditions, such as slight obstruction with moderate hepatitis

The icterus index is quite simple and measures the yellow color of blood serum in units as compared to a 1 10,000 potassium bichromate solution. It is more satisfactory than skin color which changes shade slowly. It will show an increase in jaundice when urine does not always reflect the change. The abnormal readings begin at about 15 and are said to go over 300. It is difficult for us to make what we consider accurate readings over 200. This patient's maximums were 130, 80 and 160 in the three attacks. If it changes rapidly, we may allow ourselves to think of a changing obstructive process.

The van den Bergh reaction was taken up in this country soon after the World War. The apex of exact interpretation in the effort to differentiate medical from surgical jaundice was reached about 1929 to 1932. We have come more and more to use the results of this test cautiously. In obstruction the rise from a normal reading of 1 to 3 mg rapidly rises to 15 to 30. In silent stone this does not differentiate from obstruction by neoplasm. In obstruction the immediate direct van den Bergh was supposed to be positive, the indirect positive. A delayed direct reaction was interpreted as reflecting a hemolytic form of jaundice. Where both obstructive and nonobstructive forces were supposed to be taking place simultaneously, as in toxic and infective jaundice, the biphasic reaction should occur. Jaffe's conception of so-called catarrhal jaundice being primarily a hepatitis which we believe is correct, makes such interpretations most complicated and unsound. I believe until we know more, the practitioner should use more simple criteria. In most instances the intrahepatic damage has already begun to give the delayed direct reaction by the time the doctor is able to see the patient and make the test. Our patient showed 16, 5 and 18 in the three attacks. When considered together with the icterus index, we would think perhaps that the first attack was approximately 60 per cent obstruction and 40 per cent hepatitis, the second

largely hepatitis, and the last mostly obstruction with some hepatitis. As it happened this man was about correct, but such interpretations might just as well have led us astray unless we carefully questioned and examined the patient. N. C. Gilbert has tried injecting bilirubin 1 mg per kilo into patients to study the degree of liver embarrassment. Bilirubin is very expensive and we consider this only as a "research step" in liver studies. We did not make this study in this particular case, although it should have been done.

The bromsulphalein test as described by Serby and Bloch<sup>6</sup> may be of use in the diagnosis of carcinoma of the head of the pancreas.

The blood cholesterol determinations are of some interest to those of you having technicians who make no errors. This is especially true when cholesterol esters are studied. When the liver is embarrassed the cholesterol usually removed by the liver is found increased in the blood stream. In obstruction the cholesterol goes up rapidly from the normal level of 180-200 to as much as 500 mg. If at the same time the cholesterol esters decrease disproportionately, *i. e.*, more than their 40 per cent drop to 20 per cent or less, it makes us suspect that intrahepatic damage is increasing in proportion to the obstructive process. This is clearly described by Ottenberg.

The galactose tolerance test depends on measuring the loss of efficiency of the liver as a factor in carbohydrate metabolism. After 40 Gm of galactose is given under basal metabolic conditions about 3 Gm or less comes out in the urine in five hours. If more is excreted, we may conclude that at least something is deficient in carbohydrate metabolism, perhaps the liver. This would not help us in choosing either medical or surgical management of jaundice, but it has a distinct place in metabolism clinics to further knowledge.

This patient was jaundiced for seventeen weeks in his first attack in the winter and spring of 1933. This persisted in spite of the medical management to be described later. He cleared up, felt well from June 1933 to July, 1934. He was very conscientious in his clinic visits for gastric and stool

analyses during the entire interim. He followed his diet most of the time. In July, 1934 the weather was very hot and jaundice recurred (the second attack referred to in discussion of liver function tests). This attack lasted only three weeks and was probably just a simple hepatitis with little obstruction as hinted by the icterus index and van den Bergh. All went well with him until he contracted an influenzal attack in January, 1935. He continued his work and felt very bad for a month, at which time he slowly redeveloped his jaundice. In February, 1935 he had more epigastric heaviness, but no actual pain. Thus the history of influenza suggested that hepatitis was the strongest factor, but the liver function tests pointed more to obstruction.

The many tests he had undergone were again explained to him and a third invitation for surgery was presented him. A note in the record at this time reads "If there is a neoplasm it is not a fast growing one." The diagnosis became partial obstruction of the common duct with recurring hepatitis. The patient consented and operation in March, 1935 disclosed a small stone partially obstructing the ampulla of Vater with probable mild hepatitis. The surgeon thought it inadvisable to remove a section of liver for study. There were a few very small stones in a large thickened gallbladder. The stomach and duodenum seemed normal to Dr. H. E. Jones, who performed the operation. Cholecystectomy was performed and the patient made a good recovery and has felt well since.

We may conclude from this that a hepatitis or so-called catarrhal jaundice was secondary to a partial obstruction. The liver vulnerability was probably caused by a residual infection following the first obstruction in 1928, or the partial obstruction off and on since. Difficulties arose when the patient became fatigued, during hot weather, after cold infection, or after dietary indiscretions.

At the time this patient came to the clinic in 1933 we were on our ninth year in trying a new type of management for embarrassed livers and low-grade gallbladder inflammations.

with or without stones. It may be briefly described in three stages and should be used only when medical management seems advisable after careful study.

In the presence of jaundice we follow conventional lines of absolute rest, plenty of fluids, excess carbohydrates, intravenous glucose, and rarely intravenous calcium gluconate. We are sparing in the protein allowance and begin a bland creamy diet described later as the jaundice subsides. Decholin or Epsom salts are not given in an active jaundice.

When the jaundice has subsided the first stage of management for all low grade gallbladder disorders and liver dyscrasias is gradually begun. This is virtually a strict hourly feeding ulcer management including the usual cream, eggs, cereals, custards, gelatins and butter with three pureed cooked vegetables and two pureed cooked dried fruits. If there is pain, refer to surgery. The calories are gradually increased according to the patient's tolerance to about 3000 daily. Most authorities do not believe that the bile tract empties, such as cream, butter and eggs, should be used. This clinical management follows Ivy's and Jones' idea that overcoming stasis without quite overloading the embarrassed liver's ability may help rehabilitation toward its natural physiologic function. From our studies we believe the stomach outlet is usually in difficulty at such a time and should be protected by the small, frequent, neutralizing feedings. Remember the positive ben-zidine tests in the gastric contents for the first few weeks of each attack in this patient. Antispasmodics, such as tincture of belladonna, with phenobarbital are used routinely.

The second stage of management is withheld until the patient has nearly recovered and occult blood has been absent from the stools and stomach for some time. This is the addition of trial teaspoonful doses of Epsom salt or  $\frac{1}{2}$  teaspoonful doses of magnesium oxide replacing feedings at the estimated emptying time of the stomach. The patient should never be purged, and the amount should be cut down if more than one or two bowel movements per day result. If no pain, continue the first stage of medical management. If pain results, refer

the patient to surgery This stage is as much diagnostic as therapeutic and answers the requirements for a management which will bring out latent symptoms

The third stage is used only after the jaundice has gone and the patient reverts to the chronic bile tract and liver dyscrasia classification This is the addition of decholin to the first stage treatment According to K K Jones, this markedly increases bile flow in dogs If there is pain, refer to surgery, if not, either continue on stage one or stage three

Some of the most interesting findings in this management have been the gratifying results on the gallbladder dyscrasias of which there are a surprising number These usually occur without jaundice Psychic management with antispasmodics stronger than usual are of great assistance to the patients

*The interrelation of diseases of the liver and bile tracts to duodenal disorder cannot be overemphasized* We should seek always to protect stomach and duodenal lining in any treatment for jaundice or associated disorders We should give every possible aid to smooth natural bowel peristalsis by avoiding any cathartics (except for diagnosis) The pureed cooked vegetable and dried fruits plus mucilose or metamucil can do a great deal in avoiding spasm of the bile tracts This avoids back pressure and stasis which probably helps in the rehabilitation of inflamed liver cells and bile passages

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## CLINIC OF DR JEROME HEAD

NORTHWESTERN UNIVERSITY SCHOOL OF MEDICINE

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### THE TREATMENT OF BRONCHIECTASIS

DILATATION of the bronchi was first called to the attention of Laennec by his pupil Cayol. In 1819, in the first edition of "*Traité sur l'auscultation médiate*" Laennec spoke of it as an extremely rare disease. By 1826 he had become impressed with its frequency. From that time until the present, as attention has been drawn to it and diagnostic methods have been developed, there has been a progressive recognition of its true incidence. Today it is known to stand second only to tuberculosis as a cause of chronic cough. In some hospitals it has been found in 7 per cent of all autopsies. It is probable that in this present gathering of 50 people there are 3 or 4 affected with it and I am certain that one rarely lectures on the disease to a class of medical students without having one or more come up afterward and say that they have the symptoms described.

Bronchiectasis varies greatly in the severity of the symptoms which it produces. Fortunately many cases are mild and those so afflicted lead long and active lives scarcely incommoded by their slight morning cough and expectoration. In its severer form, and the severity of the symptoms seems to bear little relation to the size of the dilatations, it is a terrible and distressing affliction, the frequent coughing spells, the foul breath and the necessity of expectorating great quantities of fetid sputum rendering the subject a social outcast. While fever, emaciation and systemic symptoms are extremely rare and most patients, even those with profuse expectorations,



appear to be in good health, the disease does threaten and shorten life. In a series of 200 cases which I studied, 50 per cent began before the tenth year, but of those over forty years old few or none dated the onset to childhood. This suggests that most of those affected in childhood succumbed to pneumonia, lung abscess, brain abscess or some of the other complications of the disease before reaching forty.

Bronchiectasis is one of the most difficult diseases to treat. Success is usually partial and often slight. The dilatations are permanent deformities which can be cured only by excision of the lung, and the chronic bronchitis is usually extremely resistant. While any one of a variety of remedies may give startling results in an occasional instance, too often all remedies fail. Today I wish to speak particularly of the treatment.

Sydenham wrote "I have come to this conviction—that those who have directed their eyes and their minds the most accurately and diligently to the phenomena of disease will excel in eliciting and applying the true indications of cure." Having in mind this practical attitude toward clinical observations, I wish today, while speaking of bronchiectasis, to concern myself not with the dilated bronchi or their forms or causes, but with the chronic bronchitis to which they predispose. Dilated bronchi neither produce symptoms, impair health nor threaten life. They do favor the establishment of chronic infection and it is this that is the true disease. Since it is the bronchitis which must be treated, a consideration of its pathogenesis—the factors which favor its inception and determine its chronicity—should point the way to logical and successful treatment.

In all of the body cavities and tracts there are four conditions which incite infection and maintain it in a chronic state. These are

- 1 Interference with the normal evacuation of secretions
- 2 Persistent reinfection
- 3 The presence of a foreign body
- 4 Infection with tubercle bacilli or other resistant organisms

In bronchiectasis the normal mechanisms for evacuation of secretions, the ciliary action of the epithelium and the peristaltic contractions of the bronchial musculature, are destroyed. These being out of function, gravity favors the accumulation and stagnation of secretions and the development of chronic infection. The role of gravity is of the greatest importance. Bronchiectasis is rare in any part of the lungs save the posterior and most dependent portions of the lower lobes. When it does occur in the upper lobes the etiology is either parenchymal fibrosis or bronchial obstruction and upper lobe dilatations are rarely complicated by chronic bronchitis or profuse secretion. These facts justify one in surmising that bronchiectasis, usually in its etiology, and practically always in its clinical manifestations, is a disease of posture—one of the many untoward results of man's assumption of the unnatural erect and supine positions. Whether or not gravity so hinders evacuation as to produce the dilatations or the diseases which produce them, it is certain that once the bronchi are dilated and the ciliary and peristaltic mechanisms compromised, gravity is the chief cause of the accumulation of secretions and the persistence of infection.

Persistent reinfection sometimes causes bronchiectasis and practically always plays a part in maintaining the infection. Chronic sinusitis is recognized as a cause of the disease and in nearly every case of bronchiectasis the sinuses are involved. In some instances they are the etiological factor and in others are secondarily infected from the continual spraying of the upper respiratory passages with the raised secretions. In all cases the chronic postnasal discharge adds constantly to the infection in the dilated tubes.

Foreign bodies, either of extraneous origin or broncholiths formed in situ, are not uncommon in bronchiectasis. When present, they are an important cause of profuse bronchial secretion.

Tubercle bacilli and other resistant organisms are probably not a factor in the chronic bronchitis of bronchiectasis. While the condition often complicates tuberculosis, chronic

His present illness started two years before with a sudden onset of profuse and repeated hemoptyses. He was sent to Edward Sanatorium where Dr George Dyche made the diagnosis of bronchiectasis and verified it by bronchography. He had remained in the sanatorium four months without further bleeding and had then returned to work. His cough disappeared entirely and he remained free from symptoms until two weeks before coming to the clinic. At this time his hemoptysis recurred and had been repeated frequently during the past few days.



Fig 134



Fig 135

Fig 134—Case I Dry, hemoptysic bronchiectasis of right lower and middle lobes before injection of iodized oil

Fig 135—Case I After injection of iodized oil

Physical examination showed nothing abnormal save decreased breath sounds and occasional coarse bronchial râles at the right base and in the axilla. x-Rays taken after the injection of iodized oil showed cylindrical dilations of the bronchi in the right middle and lower lobes (Figs 134 and 135).

On March 18, 1932 the right phrenic nerve was extracted.

Since that time the patient has had no further hemoptyses, but within the past year has developed a slight morning cough. He expectorates about  $\frac{1}{2}$  ounce of sputum each twenty-four hours.

**Case II.**—Extensive saccular bronchiectasis of left lower lobe in boy fourteen years old secondary to foreign body in nose since childhood. Marked improvement following removal of foreign body, phrenicectomy and postural drainage.

E M., Montgomery Ward Medical Clinic of Northwestern University No 45,492 This boy was fourteen years old when he first came to the clinic in January, 1931 He complained of a chronic productive cough pain in the chest, hemoptysis and nasal obstruction

His father had pulmonary tuberculosis.

His mother and 3 sisters and 2 brothers were living and well

He had measles and whooping cough in early childhood and many acute respiratory infections.

The present illness dated back to infancy The father said that the boy had always coughed and had a nasal discharge and has had repeated attacks of pneumonia For years he had coughed a great deal severe attacks occurring in the morning and whenever he exerted himself in play At these times



Fig 136

Fig 137

Fig 136.—Case II. Severe bronchiectasis of right lower lobe secondary to foreign body in nostril

Fig 137.—Case II. After injection of iodized oil.

he raised large quantities of fetid sputum. His father estimated that he expectorated more than a cupful a day Slight hemoptyses were common as were also pains in the left chest Physical examination at that time showed a poorly developed and nourished boy with profuse nasal discharge and frequent loose cough His complexion was sallow and his attitude listless

Physical examination showed moderate clubbing of the fingers and dulness decreased breath sounds and coarse bronchial râles over the lower half of the left lung

For the x ray findings see Figs 136 and 137 x Rays of the sinuses showed normal aeration

The patient was given potassium iodide and instructed in postural drainage. The left phrenic nerve was extracted

He was referred to Dr O H MacKay of the ear nose and throat depart

ment, who found a markedly deviated septum, obstruction of the right nostril and mucopus in both sides. A diagnosis of deviated septum with possible foreign body was made.

At operation Dr Maclay found a metal screw firmly embedded in scar and granulation tissue in the right nostril. The septum was straightened and the tonsils and adenoids removed.

Following these operations improvement was rapid. His cough and sputum decreased markedly and his gain in weight and general health was striking. For the past four years he has coughed only on rising in the morning and has raised less than 1 ounce of sputum each twenty-four hours. Today at the age of eighteen he is a well-developed and alert young man.

In this case it seems certain that the foreign body in the nose and the consequent nasal infection caused the bronchiectasis. That it was important in maintaining the infection in the dilated tubes is evidenced by the prompt improvement following its removal. Much elevation of the diaphragm was prevented by adhesions and it is doubtful if this operation contributed greatly to the result.

**Case III**—Severe bronchiectasis of left lower lobe, constant fever, repeated hemoptysis, treatment by medication, postural drainage, phrenicectomy and paraffin pack ineffective, cure following lobectomy.

Miss M. D., Montgomery Ward Medical Clinic of Northwestern University, No. 43,734. This patient, a girl seventeen years old, first came to the clinic in



Fig 138

Fig 139

Fig 138—Case III. Bronchiectasis of left lower lobe before injection of iodized oil. Severe and disabling symptoms.

Fig 139—Case III. After injection of iodized oil.

September, 1931 She complained of a chronic cough with profuse expectoration of two years duration The onset of her cough was gradual and there was nothing in the family or past histories which could account for it At the time that it developed she was working in a nut factory shelling nuts a fact which suggests that a foreign body may have been inhaled She was raising approximately 4 ounces of fetid sputum each twenty four hours Hemoptyses were frequent and fever was present much of the time.

Physical examination revealed a poorly developed and nourished young girl There were no abnormal physical findings in the lungs

The x ray findings are shown in Figs. 138-140



Fig 140.—Case III Following lobectomy Shadows in right lung field are artefacts

Postural drainage phrenicectomy and medication with potassium iodide were ineffective in improving her condition She grew gradually worse Her sinuses which had been normal on admission became infected In August 1932 a paraffin pack was inserted over the base of the left lung This gave no relief

In November 1933 the paraffin pack was removed and the left lower lobe resected Since that time she has gained 12 pounds in weight Her general health has improved markedly She still coughs and raises some sputum but has no fever and no hemoptyses. The operation has changed her from a chronic complaining invalid to a healthy and ambitious young woman

This case is important as demonstrating the severe and disabling type of bronchiectasis Any procedure, however radical, which offered a possibility of cure was justified It is of interest that the sinuses became infected three years after the onset of the cough while the patient was under observation

One could say much more about the details of the treatment. I would say in general that it should be intensive and radical only in proportion to the severity of the symptoms and that in the majority of cases one must be satisfied with partial results. As the mortality of lobectomy is reduced, and I feel certain that it will be, its indications in unilateral cases can be accordingly extended.

In closing I would like to emphasize the importance of cleaning up infections in the upper respiratory tract and the necessity, if this is to be accomplished, of close cooperation with the specialist in diseases of the nose. In our department we had little success from this form of treatment until all of our cases were referred to a single man. Dr. Otis H. Maclay, by frequent consultation with us, has come to understand the problem from our point of view and by so doing has been able to adjust his measures to the requirements of the particular case and to render us invaluable service.

## CLINIC OF DR VINCENT J O'CONOR

### WASHINGTON BOULEVARD HOSPITAL

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#### THERAPEUTIC VALUE OF PROSTATIC MASSAGE WITH A DISCUSSION ON PROSTATITIS AND THE SIGNIFI- CANCE OF PROPER RECTAL PALPATION OF THE PROSTATE GLAND

For discussion this morning I wish to present this patient and give a brief history of his complaint

A G., aged thirty six, came to the hospital complaining of persistent headache loss of energy and appetite. He is an unmarried clerk of sedentary habits who has always been well except for occasional sore throats and head colds each winter. He had had measles and scarlet fever in childhood and influenza in 1918. He denies gonorrhea or other urinary infection. He first consulted a physician for sacral pain two years ago. At this time an x ray was made and the patient was told that he had a "slipped sacro iliac joint." Orthopedic belts and medicine by mouth did not improve the condition. Several months later he consulted an osteopath who told him he had a "slipped vertebra" but manipulative treatments by him brought no relief. The patient has since taken frequent doses of aspirin and other sedatives with temporary relief. More recently the sacral and lower lumbar pain has been bilateral and when he tries to rise from bed in the morning the effort is most painful. During the day the pain becomes less severe and is often almost entirely absent on retiring at night. Exercise, which he has to force upon himself does not increase the pain. Other than the above, the pain is not related to bowel movements, urination or any other function. He has very occasional sexual intercourse and thinks the pain is lessened for several days after these experiences.

Physical examination reveals a well nourished man apparently in good health. The teeth are in good condition, the tonsils appear to be the seat of chronic infection. Heart, lungs blood pressure and blood count are normal. Urinalysis is negative except for a slight excess of leukocytes (10-15 per high-power field after centrifuging) in the sediment. Mobility of the spine is normal there are no areas of tenderness along the vertebrae or over the sacrum. x Rays of the teeth and spine show no abnormality. Rectal examination reveals a normal sphincter no anal fissure or hemorrhoids and a smooth rectal mucosa. The prostate is twice normal size soft and boggy without any indurations or infiltrations in the prostatic tissue. The rectal mucosa is



freely movable Both seminal vesicles are palpable and distended with a somewhat ropy consistency on outline Thorough massage and stripping of the prostate and vesicles is followed by a rapid flow of material at the external urinary meatus and this fluid on cover slip examination under the high-power field of the microscope shows 75 to 100 pus cells per field and many clumps Cultures and smears of the prostatic secretion show mixed staphylococci and colon bacilli The day following the diagnostic massage the patient stated that for the first time in two years he had no pain in his back when he awakened in the morning Our diagnosis in this case is chronic prostatitis of

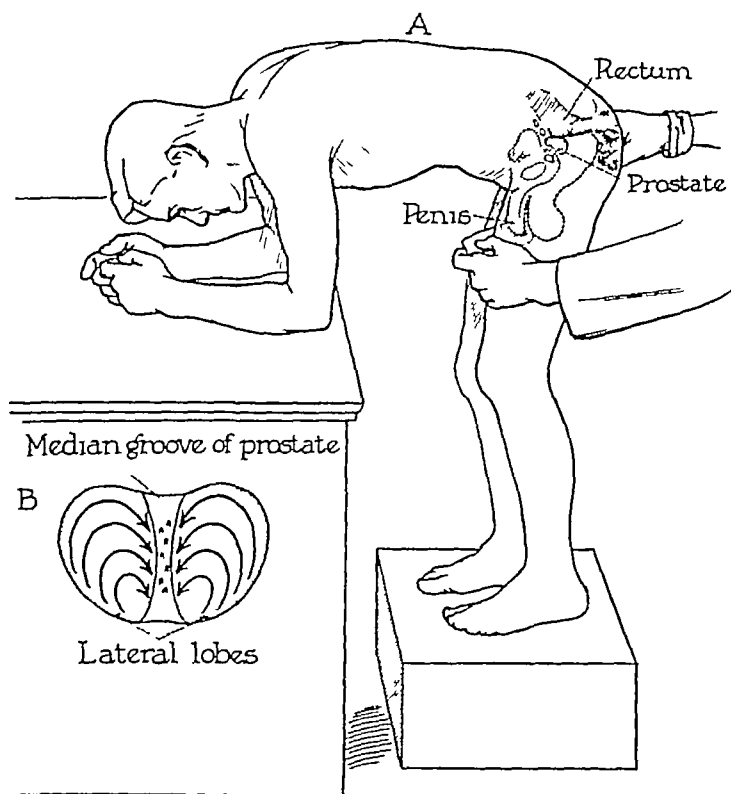


Fig 141

nonspecific origin, probably secondary to chronic tonsillitis The therapeutic procedure will include removal of the tonsils and prostatic massage at five-day intervals until the expressed material yields less than 10 pus cells per high-power field and, of course, until all symptoms are completely relieved

**Discussion**—This patient's history illustrates the necessity for a thorough rectal examination of the prostate in every male complaining of backache Unfortunately the average

physician does not include a routine rectal examination in searching for the cause of complaint in these individuals. This is an omission of which I hope you gentlemen will not be guilty. It is not necessary to be a genito-urinary specialist to make a competent examination of the prostate. The most effective examination may be made with the patient standing on a low stool or platform, the body bent over a table, toes turned in, knees slightly bent, back swayed. If the patient is bedridden, he should be examined lying upon his side with his knees drawn up. The prostate, seminal vesicles, the base of the bladder and the lower end of the ureter may be felt by the examining finger in the rectum. The finger is best protected by a rubber finger cot, using a new one for each examination. A gauze or paper shield for the hand is also discarded after the examination.

Normally the prostate as felt per rectum is heart shaped with its apex toward the anal sphincter, its base more or less notched in the center, the lateral lobes quite elastic, its central groove between the two lobes more or less marked. Normally the prostate does not project into the rectum and the lateral lobes are flat rather than bulging. Careful examination consists in sweeping the tip and flexor surface of the index finger over the surface of the gland and around its borders. All students and physicians should avail themselves of the opportunity to make frequent rectal examinations so that they may learn to differentiate minor or gross changes from the normal.

The lobes of a normal prostate are flat, flaccid and only moderately sensitive. There are no indurations or variations in consistency in the texture of the unaffected gland. Discrete hard masses felt between the rectal mucosa and the prostate proper are usually lymph nodes or phleboliths. Diffuse thickenings within the lateral lobes or projecting toward the seminal vesicles are usually of an old inflammatory nature. Carcinoma most commonly begins in the posterior lobe and the rectal mucosa becomes fixed to the gland which itself becomes immovable. Hard boardlike areas replace the normal prostatic tissue with discrete nodules of cancer to be felt in place

of the smooth, uniform normal consistency. Acute inflammations give the feeling of a tense, hot, swollen gland projecting rectally in an abnormal manner. When abscess occurs in the substance of the gland, an area of softness or fluctuation may be felt. In adenomatous hypertrophy the gland may bulge markedly into the rectum and the lateral lobes, if large, obliterate the median groove, giving one the feeling of a large, smooth, symmetrical tumor.

**Prostatic Massage**—Prostatic massage first came into popular use in Stockholm in 1894, although several references were made in Germany concerning the cure of chronic prostatitis by repeated digital expression in 1893. The technic most popular with genito-urinary surgeons is as follows.

The patient presents himself with a bladder well filled with urine and he assumes the position previously described. The protected index finger is inserted within the rectum and gentle to firm pressure made first over one lobe and then over the other with a stroking around and downward movement directed toward the openings of the prostatic ducts in the deep urethra. Finally the finger is drawn from above downward over the posterior urethra so as to express secretions from the sinus pocularis and urethral openings of the ejaculatory ducts. This manipulation should be continued for from one to three minutes. Mild massage may be indicated twice weekly, vigorous massage not more often than once a week. The therapeutic benefit derived from digital expression of the prostatic and vesicular content combines the expression of secretion and resultant improved drainage through normal channels, the stimulation of circulation in the region of the prostate proper and increased absorption from infected areas brought about by the above reactions. Rough or too vigorous massage, especially in the presence of acute infection, may force infected material down the lumen of the vas and cause epididymitis.

Prostatic massage is a useful measure in chronic catarrhal prostatitis, in chronic fibrotic prostatitis and in atrophic or atonic conditions of the prostate. It is of no value or is contra-

indicated in acute infections of the prostate, in tuberculosis, neoplasm or hypertrophy. The intelligent treatment of non gonorrheal prostatitis must include the removal of systemic foci of infection, such as abscessed teeth, infected tonsils, nasal sinusitis, etc. Stricture of the urethra, when associated must be treated by progressive dilatation of the urethra, but preferably at an interval of two to three days between prostatic manipulations. Rectal insuffusion of heat, systemic stimulation of immunity, proper hygienic advice, and urinary antiseptics may be combined to good advantage with regular prostatic massage in the treatment of chronic prostatitis.



CLINIC OF DR RICHARD J TIVNEN  
MERCY HOSPITAL

THE VALUE OF EYE SYMPTOMS IN THE DIAGNOSIS OF  
GENERAL DISEASE

**Preliminary**—The physician's greatest problem is diagnosis. His chief reliance in establishing it is the symptoms the patient presents. It is exceptional for one symptom alone—the so-called pathognomonic symptom—to determine a diagnosis. Practically always one must depend upon a group of symptoms in reaching a conclusion. Clinical experience demonstrates that the largest yield of diagnostic data results when one adopts as a routine in every case a comprehensive plan of procedure, which interrogates, so to speak, every organ or structure, which offers a possibility of contributing to the diagnostic problem. The value of eye symptoms in the diagnosis of general disease is of interest in this connection.

Eye symptomatology is of frequent aid, not alone in diagnosis of general disease, but is often also of great value in the early diagnosis and prognosis, notably in such general conditions as Bright's disease, brain tumor, arteriosclerosis, diabetes, and endocrine disturbances. A field offering such a yield of diagnostic and prognostic data should not therefore remain unexplored in general examinations. It is not unusual, however, to hear of patients who have been given what is popularly referred to as a "check up" and no investigation of the eyes has been included in this general examination. Many personal experiences might be cited demonstrating the unwisdom of such a course of procedure. I recall the case of a young child who had been given such a "check up" by a very capable pediatrician which did not include an eye investiga-

tion The little patient was given a clean bill of health and the alleged backward tendencies which prompted the examination were ascribed to the mother's solicitude and anxiety A later examination of the patient's eyes disclosed the presence of congenital cataracts Another instance of a young child whose "check-up" in which an eye investigation was omitted, resulted in the diagnosis of "gastro-intestinal disturbance," a subsequent eye examination disclosed choked disks and the diagnosis of brain tumor

Many reasons may be assigned in extenuation for the omission of an eye investigation as a routine in general examination Chief among these reasons perhaps is that the usual undergraduate medical training does not ordinarily emphasize the value of eye symptoms in the diagnosis of general disease and usually but relatively little time in the medical curriculum is devoted to training in the use of the ophthalmoscope and other ophthalmologic technic After some considerable experience in teaching in the field of ophthalmology, I hold the strong conviction that a student may with relatively little expenditure of time, training, and a negligible outlay for equipment perfect himself to a degree that he may may with confidence conduct a character of eye examination which will prove of the greatest value to his patient and of immense satisfaction to himself

As a preliminary, it is of interest to refer to an unusual phenomenon which is associated with many eye diseases Inflammation of body structures generally are usually manifested by pain, redness, edema, swelling, etc., and the function of the part is more or less impaired This does not hold true for many serious eye inflammations, especially lesions of the deeper eye structures It is quite common, for example, for a patient to be affected by such destructive eye inflammations as optic neuritis, retinitis, choroiditis and choked disk, and the external appearance of the eye be quite normal, the patient experiencing no pain whatever and his vision manifesting little or no impairment It is a grave error, therefore, to accept either the normal appearance of the eye, the lack

of the patient's complaint of impaired vision or of pain or distress of the eye as an indication that there is no active pathology present in these deeper structures. An examination of these deeper parts with the ophthalmoscope is the only safe method of discovering this hidden pathology. It is also true that many general diseases during their course attack the eye so regularly and so consistently that the eye symptomatology of these diseases should be listed among their regular general symptomatology instead of being regarded and referred to as "complications."

It may be of interest to outline briefly the various tests and technic used in conducting an eye investigation.

### EXAMINATION OF EYES

**1 Eye History**—A special eye history should always be obtained in addition to the general history. If glasses are worn, it should be ascertained why they were ordered and the results of their use.

**2 Inspection of the Eyes**—This is the first test made. The patient is placed in good daylight illumination and a general survey is made of his eyes. The main features to be noted are first, changes affecting the eye appendages, lids, tear channels, etc., such as edema or inflammation of lids, drooping of lids (ptosis), inability to close lids, arrangement of eyelashes, eye discharge, lacrimation, inspection of lacrimal puncta, tear ducts and lacrimal sac, eversion of lids to determine condition of conjunctiva, photophobia, next the eyeball itself—if it be reddened, its position, if prominent (exophthalmos) or sunken (enophthalmos), or if one eye deviates (strabismus, paralysis, etc.), finally testing for gross impairment of globe movements is made by holding a finger in front of patient's eyes and directing him while keeping head still to follow its movements in various directions, if binocular movement is normal both eyes should follow equally without lagging the movement of the finger in all directions, also the testing the lid movements, openings and closure, including the various lid signs detailed under Exophthalmic Goiter. The



examination of the cornea, pupils, etc., is best carried out with special tests to be referred to later

3 **Test of Vision (Visual Acuity)**—In all tests of vision, each eye is tested separately and the eye not being tested is covered, but not pressed down. If glasses are worn, vision should be taken without and with them. Vision is tested for *distance* and for *near* (reading distance)

*Test for Distance Vision*—For this purpose a large card upon which is printed letters of various sizes arranged in lines

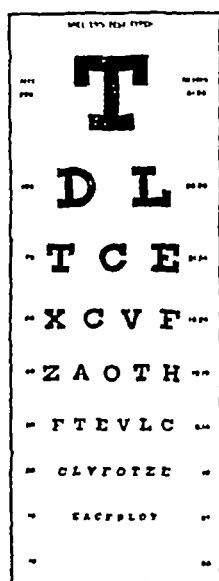


Fig 142—Test of vision Tests for distant vision—Snellen's test types

is used, called *Snellen test types* (Fig 142). Each line of letters is marked by a number above the line. The card is hung on the wall at a distance of 20 feet from the patient and on a level with his eyes, care being taken that the card is well illuminated. The distance of 20 feet is determined upon because rays of light emanating from objects at that distance are practically parallel and a normal eye requires no accommodation effort in bringing parallel rays to a focus, it is in a state of accommodative rest. The line marked 20 is therefore accepted as the standard of normal distant vision. If the

patient can read all the letters of this line at a distance of 20 feet, his vision is normal and it is recorded as a fraction  $\frac{20}{20}$ , the numerator indicating the distance the patient is from the card, the denominator the number of the line read.

Should the patient be unable to read the normal vision line—20—, he reads then the smallest line of letters he is able and the result is recorded similarly by the fraction as above. If he is unable to read the top line of letters on the chart marked 200, he is directed to approach the chart and stop when he is able to read the top line, his new distance from the chart is then estimated and if it be, for instance, 10 feet, his vision is recorded as  $\frac{10}{200}$ , the numerator—10—indicating the new distance he is from the card, the denominator—200—the line he is able to read. If he cannot read the top line marked 200 at any distance, he is directed to count fingers held a short distance in front of eye and the result is recorded as for example, "Counts fingers at 2 feet". Should he not be able to count fingers, his perception and projection of light is tested. This test is made in a dark room, the patient seated, an electric wall or floor light being behind him. He is directed to look directly forward and hold his eye perfectly still throughout the test. The examiner stands directly in front of the patient and with his ophthalmoscope reflects light into the patient's eyes from various angles, aiming in so doing to test the functional integrity of the center and the extremes of the retinal periphery. The patient is directed to say "now" the instant he sees the light, this indicates his *light perception*, then he is directed to reach out with his hand and touch the source of the light (the ophthalmoscope), this indicates his *light projection*. If he is wholly unable to "perceive" the light, the eye is blind. If he is able to perceive "the light" but is unable to "project" it (locate its source) accurately, this result is recorded as "perception—present, projection—poor" (or other qualifying phrase that clearly indicates the condition).

Test types are also usually marked with the metric scale, 6 meters being the equivalent of 20 feet and "u" being the

equivalent of  $\frac{2}{20}$ . Most cards also state after each line the percentage of normal vision the line represents. Where a distance of 20 feet is not available it may be obtained by the use of a mirror, the test type being placed behind the patient and the letters reflected into the mirror hung on the wall opposite the patient. There is also a visual testing apparatus on the market which enables one to test vision at any distance. When the patient is an illiterate, a card is used with the letter E placed in different positions, the patient being

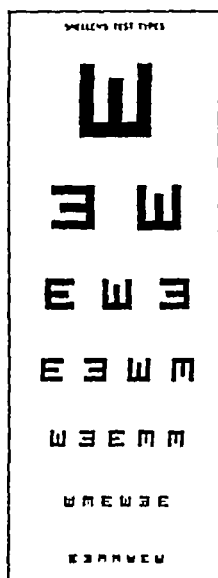


Fig 143 —Test types for illiterates (May, "Diseases of the Eye," Courtesy of William Wood and Co)

directed to indicate by his hand the direction of the arms of the E (Fig 143). The symbols for recording vision may be either those of the Latin words, "*oculus dexter*" abbreviated to the symbols, O D, or its English equivalent, R E (right eye) and "*oculus sinister*" abbreviated to the symbols, O S, or its English equivalent, L E (left eye) or "*oculus uterque*" abbreviated to the symbols O U, or its English equivalent, B E (each or both eyes). The complete formula for recording the vision should, for example, read

*Vision*OD (or R.E.)  $20/_{40}$ OS (or L.E.)  $20/_{40}$ 

*Test for Near Vision*—For this test a small printed card upon which is printed a series of paragraphs in different size type is used, called *Jaeger's test types* (Fig 144) Each

**No 1**

Engaged in manual occupation of some sort, the  
 laborer has little opportunity either to try or to release his  
 eyes of strain; his sight, under constant strain, becomes

**No 2**

many diseases or the consequences of constitu-  
 tional disorders, remains good, though the  
 acuteness lacks that strains development

**No 3.**

which follows abundant use in  
 higher types of occupation. But  
 with the literary worker it is differ

**No 4.**

ent, keeping pace more or less  
 with mental activity the eye  
 is constantly called upon for

**No 5**

action in reading for infor-  
 mation and reference on the  
 one hand in recording the

**No 6**

fruits of such occupation  
 on the other. Observation  
 has shown that deteriora

**No 7**

tion in eyesight and  
 changes in the form and  
 hence in the dioptric

Fig 144.—Test for near vision—Jaeger's test types. (May 'Diseases of the Eye," Courtesy of William Wood and Co)

paragraph is numbered. The patient with the card held at the usual reading distance (10 to 13 inches) is directed to read the smallest type paragraph he is able to read and the result is recorded for example, as Reading = Jaeger—No 2

4 **Visual Field Tests for Form and Color**—A satisfactory method of disclosing gross changes in the visual form field is as follows. Patient and examiner are seated near to and exactly opposite each other. To test the patient's right

eye for a field of vision, the patient covers his left eye, the examiner his right eye, each looks directly and steadily at the other's open eye throughout the test. The examiner then extends his left arm, with fingers spread apart, to his extreme left in the horizontal plane and slowly brings the fingers inward midway between the patient and himself. The patient is directed to say "now" the instant he sees the advancing fingers. The examiner uses his own recognition of the advancing fingers to check the patient's responses, a marked discrepancy between the two is evidence of impairment of the visual field in the meridian tested, in this case the temporal field. The nasal field is similarly tested by the examiner extending his right arm and slowly advancing them to his left with fingers spread to his extreme right, likewise the upper and lower fields by alternately advancing the fingers from the extreme upward position, downward and then from the extreme downward position, upward, the directions to the patient being the same as in the first instance. The patient's left eye form field of vision is tested in the same way, the patient covering his right eye, the examiner his left. The color fields also may be tested in a similar way using small squares of colored paper suspended between the points of a writing pen. For more refined tests of the visual fields it is necessary to employ the perimeter, campimeter or the various screens.

**5 Testing the Tension of the Eye**—This is quite satisfactorily done with the fingers. The patient is directed to look downward and to avoid "squeezing" the lids. The examiner then places the tips of the index finger of each hand upon the closed upper eyelid approximately just back of the corneal periphery and gently dimples or palpates the underlying eyeball and thus obtains information of its degree of hardness or softness. The tension of the fellow eye is the standard for comparison (Fig 145). The symbols used for recording the findings are T= meaning tension, the letter N= meaning, normal, and the signs plus + and minus —, used as follows

T=n = normal tension

T+1 = appreciable hardness

T+2 = decided hardness

T+3 = stony hardness

T- = diminished tension

T-1 = appreciable softness

T-2 = decided softness

T-3 = very soft

For more exact tension estimates the tonometer is used

#### 6 Oblique Illumination Test (Lateral or Focal) —

This test supplies information of eye structures which occupy



Fig 145—Testing of tension (May "Diseases of the Eye" Courtesy of William Wood and Co)

the area from the cornea to the depth of the anterior portions of the vitreous, viz—the cornea, the anterior chamber, the iris, the pupil, the lens and anterior portion of vitreous. Dilating the pupil will enable one to study more minutely the presence of iris adhesions to the anterior capsule of the lens (anterior synechiae), the lens and the forward portion of the vitreous. For more accurate examination of the lens and vitreous, however, the *direct illumination test* is necessary. The oblique illumination test requires 2 convex lenses of 2-3 inch focus, it is more convenient to use a loupe in place of

the magnifying lens The test should be conducted in a darkened or better a dark room (Fig 146) The light is placed approximately 18 inches to the side of and slightly in front of the patient and on a level with his eyes The examiner adjusts the loupe to his own eyes, holds the focusing lens at its rim with the thumb and index finger, applies his little finger to the patient's face to steady it, and concentrates the light on the patient's cornea The focusing lens should not be tilted, but its surface should be at right angles to the light For examination of the cornea, anterior chamber and iris, pupil and anterior portion of lens, the light is concentrated

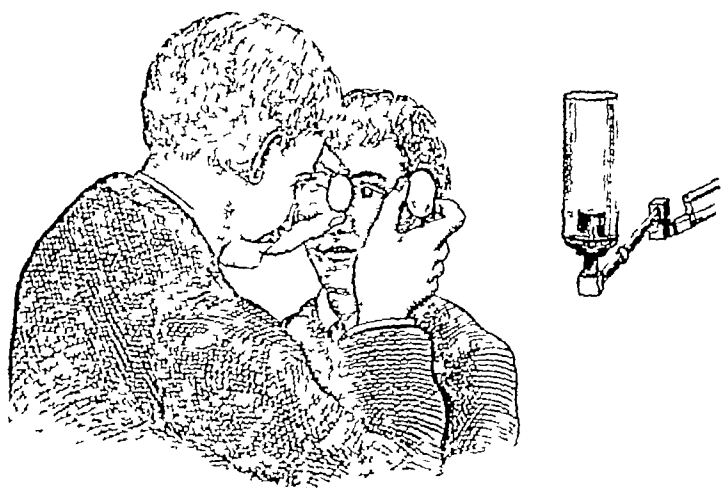


Fig 146—Method of oblique illumination (deSchweinitz)

on these structures in an oblique direction, for investigation of the central and posterior areas of the lens and forward portions of the vitreous its incidence should approximate the perpendicular For identification of minute foreign bodies in the cornea, iris or lens or fine opacities of cornea or lens a lessened illumination or utilizing the edge of the concentrated light is more useful The reactions of the pupil may also be investigated with this test, but they are perhaps better included in the special test of the pupils

**7 Pupil Tests**—These tests should be carried out in a dark room They determine the pupil equality, its regularity

of outline, size and reactions. The reaction tests are three, direct light, consensual and accommodation convergence. The *direct light reaction* is made by alternately reflecting light—either with the focusing lens or the ophthalmoscope on and off the pupil and noting its response, the *consensual* reaction is tested similarly, except the reaction of the unilluminated eye is observed, the *accommodative convergence reaction* is obtained by holding the fingers in the midline close to the eyes and noting both the convergence of both eyes and the associated pupillary contractions, as the fingers are brought



Fig 147—Direct illumination (Curt Adams)

close to the eyes. The clinical value of these pupillary tests is important and they should be made as a routine in all examinations.

**8 Direct Illumination Test**—This test is made by reflecting light into the eye with the ophthalmoscope used either with the aperture or any convex lens desired, held approximately a foot from the patient's eye (Fig 147). It is used to ascertain the condition of the media—the cornea, aqueous, lens and vitreous, opacities in these appearing as black or dark spots, if the patient be requested to move the eye quickly



in various directions and suddenly halt its movement, floating opacities in the vitreous, as dark spots, will float by the illuminated pupil

**9 Ophthalmoscopic Test**—A good electric ophthalmoscope is required (Fig 148) The direct method is satisfactory for the usual examination, it gives a magnification of 15 diameters and presents the eye structures in the upright or natural position It is usually necessary to dilate the pupil to obtain a satisfactory view For this purpose either cocaine—4 per cent solution or euphtalmine—2 per cent solution or homatropine—2 per cent solution is used, 2 to 3 drops of



Fig 148—Use of ophthalmoscope—direct method (May, "Diseases of the Eye," Courtesy of William Wood and Co)

solution instilled at five-minute intervals being usually sufficient, the eyes remaining closed during the period of instillation to avoid drying the cornea The tension should always be taken before the mydriatic is used and if it is elevated a mydriatic should be omitted Following the use of the mydriatic, as a routine, 2 or 3 drops of eserine sulphate,  $\frac{1}{4}$  per cent solution, should be instilled at five-minute intervals to secure contraction of the pupil as a precaution against inaugurating a glaucoma

**Examination of the Eyegrounds**—There are four elements one must identify in examining the eyegrounds The

*optic disk* (optic papilla), the *macula*, the *retinal vessels* and the *general fundus* (Fig 148a) Obtaining simply a general or "panorama" view of the eyegrounds as a whole is not sufficient Each of the four elements or components above mentioned must be studied individually

*The Optic Disk*—This is the first element identified, it is a "landmark," so to speak, it is the point of entrance of

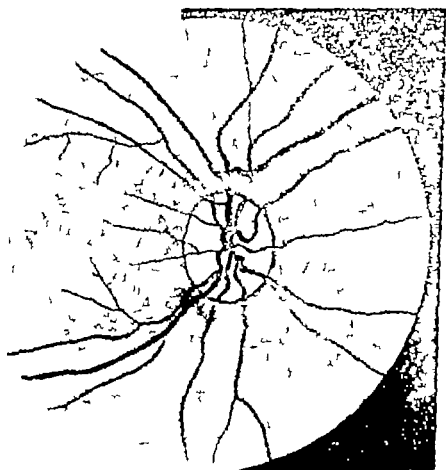


Fig. 148a—Normal fundus of the uniform stippled type The papilla is oval, has sharply defined margins is normal in color the pigment epithelium is concentrated about the papilla and in the region of the macula. The dark larger vessels without distinct light streaks are the veins the bright narrower ones with distinct light streaks are the arteries (Adam Foster "Ophthalmic Diagnosis.")

the optic nerve in the eye and hence is often referred to as the nerve head, it is also the physiological "blindspot", the disk is located slightly nasal to the posterior pole of the eye, its form is circular or oval pale pink in color, though this varies in degree depending upon the pigmentation of the gen

eral fundus, is usually paler on its temporal side, its margins are well defined and two rings usually surround it—a white inner ring, the scleral, and an outer dark ring, the choroidal, on its temporal side a funnel-shaped excavation is commonly seen, the vascular funnel or physiological cup, caused by the separation of the nerve fibers, at the bottom of which may be seen the stippled gray dots of the lamina cribrosa (Fig. 149). The chief features concerning the optic disk to be noted are

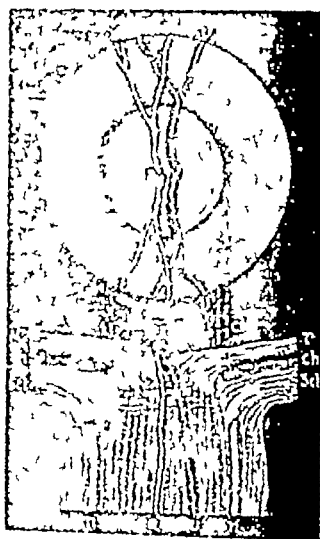


Fig. 149—Ophthalmoscopic view and longitudinal section of the disk. *a*, Central artery, *v*, central vein, *E*, physiological excavation, *s*, scleral ring, *c*, choroidal ring, *r*, retina, *cl*, choroid, *sc*, sclera (May, "Diseases of the Eye," courtesy of William Wood and Co.)

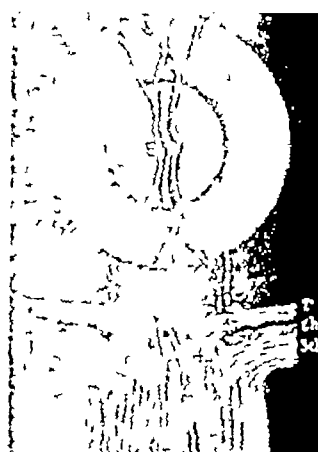
its color, the definition of its margins, changes in its surface and its level (swollen, etc.) compared with surrounding retina.

The macula (*macula lutea*—yellow spot) is located approximately two disk diameters to the temporal side and a little below the horizontal meridian, it is identified as a rather oval ring, its long axis horizontal, a bright red spot at its center is the fovea centralis. Slight pathologic changes at the macula interfere seriously with vision—it therefore should be inspected with exceeding thoroughness.

*The Retinal Vessels*—At the disk the central vessels, artery and vein, divide into a main upper and lower branch and these again branch into temporal and nasal branches, the arteries are smaller, redder, run a straighter course, and their light streak is more defined than the veins, the veins are larger, darker, course more tortuous, light streak not so defined as the arteries, arterial pulsation is uncommon and probably pathologic, venous pulsation especially on the disk is not infrequent and is probably not pathologic there is no consistency in the matter of vessel crossings, a vein may cross an artery or an artery a vein, an artery, however never crosses an artery, nor a vein, a vein. The light streak of the arteries divides the vessel into two red lines, normally the walls of the vessel itself are transparent and cannot be seen, hence the red lines observed represent the column of blood in the vessel and not its walls. The important features of the retinal vessel to be noted are, the relative size of arteries and veins, variation in their caliber, undue tortuosity, changes in their walls, interruptions in the blood current, hemorrhages along their courses, and pulsations.

*The General Fundus*—The general appearance of the fundus is due to the red reflex of the vessels in the choroid and this appearance varies depending upon the amount of pigment in the choroid itself and in the retinal pigment layer. Examination of the eyegrounds of blondes, brunettes, negro, mulatto and albino will familiarize one with these varied appearances.

The above described eye tests are definitely within the scope of the general physician and may be carried out during the general examination with a relatively small expenditure of time and effort. Necessarily the interpretation and clinical application of the findings will require some further study and investigation. This may be done by consulting one of the many excellent small volumes on medical ophthalmoscopy, supplemented by practice in the use of the ophthalmoscope on the schematic eye, equipped with colored disks illustrating the normal and pathological eyegrounds.



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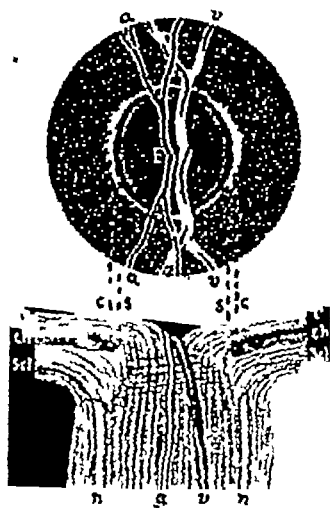


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We will now discuss more or less briefly a few diseases in which eye symptomatology is a rather frequent feature, which will serve to emphasize the diagnostic value of eye symptomatology in general disease

#### HEADACHES—EYESTRAIN

Headaches are one of the most frequent and distressing maladies which usually come first for relief to the general physician. Very many headaches may properly be classified as eye symptoms, especially those chargeable to "eyestrain." These so-called "eyestrain" headaches are due to a variety of ocular causes, such as refractive errors, particularly hypermetropia, low grades of astigmatism, fatigue of the ciliary muscle, muscular imbalance, etc.

The recognition of the possibility of headache being due to eyestrain is, therefore, a matter of interest and concern to the general physician. There are a number of special features commonly associated with eyestrain headache which strongly suggest the likelihood of the eyestrain being the cause of the head distress. These special features may be briefly summarized as follows:

The *location* of the headache is not always as one might expect a helpful diagnostic aid, any area of the head may be attacked, it is, however, quite usual to have it referred to the region of the eyes—mid, supraorbital, or frontal, and quite commonly to the region of the occiput, headaches provoked by use of the eyes, notably unusual eye demands, such as "shopping," "theater," "motoring," "embroidering," are of especial significance, the time of onset, while suggestive, is not a dependable factor, even the so-called "waking headache" which appears after a good night's rest is often found to be an "eyestrain" headache, due to the fact that on awakening the accommodative function is called into instant and abrupt use and it is this accommodative strain and fatigue which plays the major rôle in headache due to eyestrain. Other suggestive factors are such "asthenopic symptoms" as blurring of vision, irritation and burning of eyes, spots before eyes,

redness of lids, blinking and lacrimation, photophobia, inability to continue sustained use of the eyes without a feeling of fatigue or strain. Very frequently sufferers from low degrees of refractive errors complain of symptoms such as vague, ill-defined headaches which completely disguise the origin of their difficulty as being due to eyestrain. A characteristic of this class of cases is that seldom do they associate their headaches and other symptoms with their eyes and few of them have impaired vision. There are other very serious eye and general diseases in which headache is commonly an outstanding symptom. One of the most destructive of eye diseases, viz., acute inflammatory glaucoma, is the cause of severe headaches and since its tendency is to rapid involvement of sight, its early recognition is paramount for conservation of vision. Fortunately, as a rule, in this disease suggestive eye symptoms, such as foggy vision, redness of the eyeball, pain in the eye itself, dilatation of the pupil, and especially increase of the intraocular tension are associated with the headache and direct one's attention to the eye as being its cause. It should be noted, however, that often in the early stages of glaucoma, the so-called "prodromal stage," these suggestive eye symptoms are often absent. These "prodromal types" should be kept strongly in mind, they are frequently only diagnosed by a very searching eye investigation. In the other variety of glaucoma—the noninflammatory type, the so-called chronic simple glaucoma—the patient may not complain of severe headache, but only, perhaps, of a vague, rather ill-defined head or eye distress and the eye exhibits no inflammatory signs, but is normal in appearance. This type of glaucoma is an insidious, destructive eye disease, common in elderly people and requires, as a rule, thorough repeated eye examinations to exclude it.

Headache usually of a severe type is also often an outstanding symptom of brain tumor. The familiar symptom triad of headache, emesis and vertigo should always arouse a suspicion of brain tumor. Examination of the eyegrounds should always be done in such cases, it frequently discloses

the characteristic eye symptoms of brain tumor, viz, optic neuritis or choked disk.

**Practical Eye Diagnostic Summary.**—In all patients suffering with headaches of any form, severe, vague or ill-defined, the eyes should be investigated. The presence of normal vision, the normal appearance of eyes as well as absence of complaint of direct eye symptoms, does not rule out the possibility of the eyes being accountable. In elderly patients, particularly, glaucoma is the important eye disease to exclude and since increase of tension is the outstanding symptom of glaucoma, the tension should be taken as a routine in all patients. Low grade refractive errors, astigmatism, hypermetropia, are the cause of a large number of headaches and therefore the refractive condition of the eyes should be determined. A persistent headache should always suggest brain tumor, and an eyeground examination should be made for confirmation.

#### EXOPHTHALMIC GOITER

(Parry's Disease Graves' Disease Basedow's Disease)

A great number of eye symptoms or signs have been described as associated with this disease. Among them the following may be enumerated: Exophthalmos, von Graefe's, Dalrymple's, Stellwag's, Moebius', Kocher's, Gifford's, Suker's, Jellinek-Teillais', ophthalmoplegia, etc. Of these, there are six symptoms, exophthalmos, von Graefe, Dalrymple, Stellwag, Moebius, Gifford, which are relatively constant and are commonly accepted as being a part of the symptom complex of the disease. For clinical purposes, and without regard for the various theories assigned as causal factors in their production, I have found it of advantage to group these seven symptoms or signs in the following manner. *One* of them, the *exophthalmos*, concerns the *position* of the eyeball, *four*, the *von Graefe, Dalrymple, Stellwag, and Gifford*, concern the *cyclids*, *one*, the *Moebius*, concerns the *movement* of the eye ball (convergence, etc.) These may be discussed briefly as follows:

**Exophthalmos**—Exophthalmos, when apparent, is the most striking symptom of Graves' disease and is the symptom from which the term, exophthalmic goiter, usually applied to the disease originated.

In the earlier studies of the disease the triad of symptoms—exophthalmos, tachycardia and thyroid enlargement—were regarded as the “diagnostic syndrome” of Graves' disease. More recent clinical research has altered and amplified this concept. The symptom of exophthalmos, however, is still regarded, with such reservations as these later researches have suggested, as an important part of the symptomatology of the disease. Formerly, it was believed that exophthalmos was an invariable accompaniment of the disease symptomatology. At present it is said to be absent in atypical cases—the so-called *formes frustes* types. Bram believes that “exophthalmos occurs sooner or later in from 65 to 85 per cent of subjects of Graves' disease.”

In addition to the term, “exophthalmos,” the symptom—depending somewhat upon its degree—is often described as a “bulging” or “prominence” of the eyes, also as “pop-eyes,” “goggle eyes,” “proptosis,” etc.

The malposition of the eyes is straight forward, the degree of prominence varying from slight protrusion, at times difficult to estimate, to extreme protrusion interfering with lid closure, the latter condition exposing the eye to the grave danger of corneal ulceration, panophthalmitis, possibly loss of the eye.

The mobility of the eye is rarely impaired to any considerable degree. These two characteristics—the straight forward proptosis and the retention of practically normal mobility of the eyeball, are of considerable value in differentiating from an exophthalmos due to orbital or nasal accessory sinus pathology.

Both eyes are usually involved. A difference in degree between the two eyes is frequent, notably in the early stages of the disease, only one eye may also be affected, the other appearing normal.

thalmos Reports as to the incidence of the symptom vary from 15 to 55 per cent

Sharkey (referred to by Foster Moore) states that the symptom is not limited to Graves' disease but that of 613 cases of all kinds of disease, excluding Graves', it was present in 12 cases and that many people can produce it voluntarily.

Willebrand and Saenger (referred to by Dock) give five theories of the cause of the symptom, viz Sympathetic, central, action of the orbital vessels upon the levator, insufficiency of the orbicularis, increase of the forces which cause elevation

**Dalrymple's Sign**—This sign consists of a retraction of the upper lid with consequent widening of the palpebral fissure, the sclera being exposed above and below the cornea. This produces in the patient the so-called "*look of fright*" or "*staring expression*" According to Dock, "it occurs early, is rarely absent, varies from day to day and sometimes is more distinct on one side" This sign is also found in hysteria, tetanus, pregnancy, and in maniacal maladies. Its cause is attributed to spastic contraction of the musculus tarsalis superior due to irritation of the sympathetic (Fuchs)

**Stellwag's Sign**—This sign consists of a diminished frequency of winking. Normally the function of winking distributes the lacrimal fluid over the cornea, bathes it, clears the cornea of dust and prevents the corneal layers from drying. Winking takes place normally, approximately three to ten times per minute, in Graves' disease it may not take place more than once in several minutes. Interference with this corneal protective function exposes it to irritation and infection. It is not uncommonly an early symptom and is said to occur in 30 to 50 per cent of cases.

**Moebius' Sign**—This sign is a reduction in the convergence function not attributable to muscular paralysis or exophthalmos.

**Gifford's Sign**—In this sign difficulty is experienced in evertng the eyelid, and is regarded as of frequent occurrence.

The foregoing résumé of the ocular manifestations of

Graves' disease emphasizes the value of these symptoms to the clinician as aids in the diagnosis and in estimating the clinical progress of the disease

**Practical Eye Diagnostic Summary**—In all cases of suspected exophthalmic goiter the eye and its appendages should be examined. The eye symptoms exhibited concern mainly the position of the eyeball, lid movement and convergence. The various impairments of these—referred to—should be investigated in a detailed way in every case.

### ARTERIOSCLEROSIS

In November 1931 a patient aged fifty nine, consulted me for change in reading glasses. no general complaints. considered himself to be in excellent physical condition. Examination of the eyegrounds disclosed an arteriosclerosis of the retinal vessels of marked degree. He was advised to have a general physical examination. Within the following four months the patient was given a thorough general physical examination in two different outstandingly recognized clinics. The findings of both clinics, other than hypertension of moderate degree were negative. Approximately five months later the patient while attending a hockey game was stricken with cerebral hemorrhage from which he later died.

According to Fraser's survey of life insurance mortality experience for the year 1934, "the mortality from the cardiovascular renal group (a group that includes heart disease, chronic kidney disease, apoplexy, and arteriosclerosis) far outweigh those from any other cause."

The sequential relationship of hypertension, arteriosclerosis, the cardiovascular-renal group of diseases, and vascular cerebral pathology, is, as yet, undetermined. Whatever the final verdict may be, it is exceedingly probable that arteriosclerosis will occupy a prominent place in the final estimate. As Osler well says, "the tragedies of life are largely arterial," and one may add, arteriosclerosis plays a major rôle in their enactment. Early recognition of a beginning arteriosclerosis is therefore of outstanding value in conserving life. The retinal vessels, in common with other general vascular structures share in the attack of sclerotic processes, often they present evidences of such invasion before it is manifested in other portions of the body. It is not too much to say that no part of the body offers a more fruitful field and relatively easy

method of obtaining reliable data concerning the condition of vascular structures than that offered by the retinal vessels and fundus. Numerous observers confirm this view.

Osler, for example, states, "Of all the vessels in which to see early thickening, the retinal arteries are the most impor-

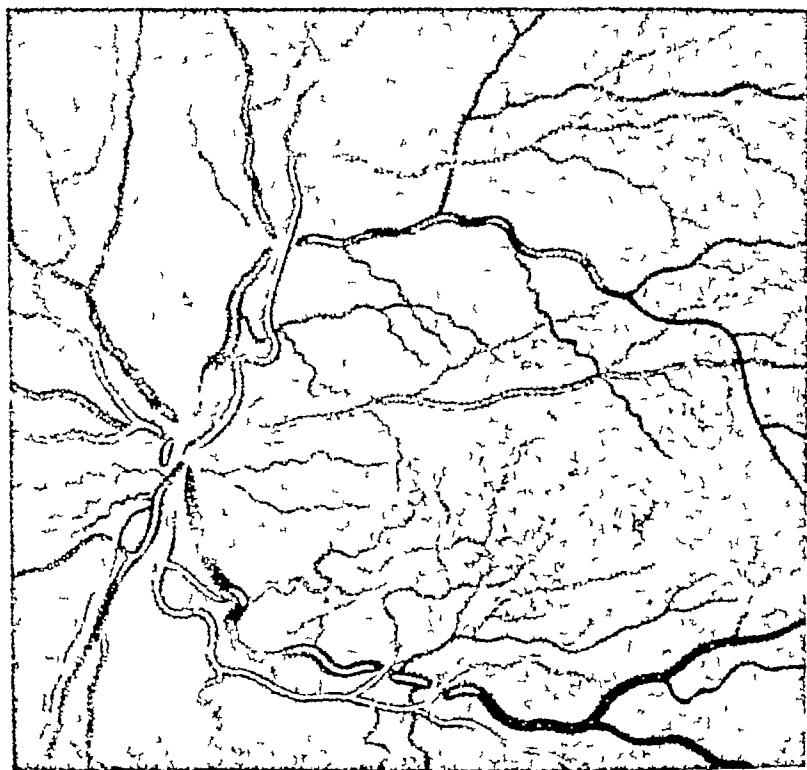


Fig 150—A case of arteriosclerotic retinitis. From a woman aged sixty-five, whose systolic blood pressure was greater than 300 mm. She died of "a stroke totally blind" four years and four months after the drawing was made. The arteries are very bright, the lower one showing a dotted reflex, and when seen three years and one month later many of them were converted into fibrous threads. The veins are deeply cut into by the arteries, in one place a vein rides over a thickened artery. A few hemorrhages and a number of the discrete dots of arteriosclerotic retinitis are seen. The disk edges are blurred. (Moore, "Medical Ophthalmology," J. and A. Churchill, Publisher.)

tant." Gunn observes, "ophthalmoscopic examination is one of the most ready clinical means for the early detection of important arterial changes," and Friedenwald notes, "the

most important aspect of ocular and especially retinal arteriosclerosis is its diagnostic value in relation to general vascular disease" Cabot writes, "arteriosclerosis may appear earlier or more clearly in the retina than elsewhere"

The pathologic histology of retinal arteriosclerosis is of interest in interpreting the varied symptomatology Adams' view is, "microscopically, the process is seen to be due to a chronic inflammatory process, there being a proliferation of the endothelium of the tunica intima, and at the same time new formation of connective tissue and especially of elastic tissue The endothelial proliferation occurs both in the arteries and veins" The obstruction of the retinal vessels, which those various lesions produce, affects the nutrition of the retinal structures and is therefore a major factor in impaired retinal function

The arteriosclerotic changes which affect the retina and retinal vessels are many and various The following abridged outline of deSchweinitz' comprehensive summary presents the usual changes encountered (Fig 150)

"Alterations in the retinal vessels are also caused by chronic nephritis and general arteriosclerosis and present the following ophthalmoscopic appearances (1) Alterations in the course and caliber of the retinal arteries, such as undue tortuosity, which is not significant unless it is associated with other evidence of disease, in the size and breadth of the retinal arteries, presenting as it were a beaded appearance (2) Alterations in the reflections from and the translucency of the walls of the retinal arteries, manifesting themselves (a) in increased distinctness of the central light streak on the retinal vessel and an unusually light color of the entire breadth of the artery, (b) loss of translucency, so that it is impossible to see, as is possible in the normal state, through the artery and underlying vein at the point of crossing, (c) positive changes in the arterial walls consisting of whitish stripes, indicating degeneration of the walls or infiltration of the perivascular lymph sheaths (perivasculitis) (3) Alteration in the course and caliber of the veins, together with signs of



mechanical pressure, manifesting themselves (a) in undue tortuosity, which as in the case of the arteries is not significant except in the presence of other disease, (b) alternate contractions and dilatations, (c) an impeded venous circulation where a diseased artery crosses it. The last is a sign of the utmost importance. (d) Changes in the venous walls, precisely as they occur in the arteries, so that whitish stripes border the vessel and are indications of degeneration in its walls. Often associated with this one may see varicosities. (4) Edema of the retina, manifesting itself (a) as a grayish opacity, which may be present in the immediate neighborhood of the papilla, or in spots over the eyeground and along the course of the vessels, looking like a fine gray haze or in little fluffy islands far out in the periphery. (5) Hemorrhages, manifesting themselves as linear extravasations along the course of the vessels, roundish infiltrations scattered over the fundus, or in droplike form."

He further adds, "the *significance* of these lesions is of serious import. In addition to their relation to nephritis, they may be the forerunners of vascular sclerosis of the brain or indicate the presence of disease of the cerebral arteries. Their subjects are liable to hemorrhage in the brain and all its consequences." The consensus of opinion is that no one sign or symptom in the retinal vessels or retina is pathognomonic of retinal arteriosclerosis. Moore, for example, points out that tortuosity of the arteries of itself is not a reliable index, deSchweinitz emphasizes, however, that a well marked tortuosity affecting the smaller vessels of the macula, indicates a marked degree of arteriosclerosis.

Optic atrophy of gradual development may supervene as a result of the reduced blood supply occasioned by the sclerotic vascular disease. Oatman calls attention to a pulsation in the arteries, so-called "locomotion pulse" as one of the early evidences of retinal arteriosclerosis. He emphasizes as a diagnostic aid the pulsation pressure test, viz. "Healthy retinal vessels are very compressible while sclerosed vessels are comparatively incompressible. If gradually increased pressure is

applied by the finger to the eye the ophthalmoscope will disclose (1) strong venous pulsation on the disk, (2) arterial pulsation, (3) cessation of circulation and blanching of the vessels. Rigid vessels resist pressure and where retinal arteriosclerosis is advanced neither venous pulsation nor blanching of the vessels can be produced by pressing on the globe with the finger."

Other suggestive symptoms of arteriosclerosis are spontaneous subconjunctival hemorrhages in adults, edema of the lids and recurring conjunctival chemosis. Moore and many other observers stress the diagnostic value of the peculiar phenomena occurring at the arteriovenous crossings, such as indentations and obscuration of the underlying vessel, diver

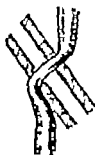


Fig 151.—To show the riding of a vein over a thickened artery in the retina. The shaded band on each side of the artery indicates its visible coats. From a man of sixty five whose systolic blood pressure was 265 mm. The appearances were quite unchanged at the end of two years. (Moore, "Medical Ophthalmology," J and A Churchill Publisher.)

sion of its course and interference with its visibility for short distances on either side of the crossing, these changes he regards as evidence of a high degree of sclerosis (Fig 151). Knapp emphasizes that in appraising the ophthalmoscopic findings, refractive errors, conditions of acute toxemia occurring in acute Bright's disease, severe influenza, and a failing heart must be excluded. Thorington mentions the presence of sclerosis in retinitis pigmentosa. Cerebral arteriosclerosis is not necessarily to be predicated on a retinal arteriosclerosis as the two may not coexist. Herter, however, states that a positive finding of sclerosis of the retinal vessels assumes a similar condition in the cerebral vessels, but not the reverse.

Moore believes that where disease of the retinal arteries is evident, cerebral vascular disease is also present, but that in 30 per cent of cases even an advanced disease of the cerebral vessels may be present without the retinal vessels being demonstrably involved. As to prognosis, Moore states the tenure of life of patients who are the subjects of retinal arteriosclerosis is uncertain, but a considerable number live for several or many years. Adams concludes that the prognosis is dependent upon the associated renal and cardiac changes and that it is aggravated by the presence of albuminuria and that it is better in older people.

**Practical Eye Diagnostic Summary**—Early manifestations of general arteriosclerosis are frequently seen in retinal vessels. Their early recognition have an important bearing in relation to the cardiovascular-renal group of diseases. The presence of sclerotic changes in the retinal vessels suggests arteriosclerosis, syphilis, or nephritis. Routine examination of the eyegrounds is therefore of outstanding value in general examination.

The important changes to be sought for in ophthalmoscopic examination of the retinal vessels are—undue tortuosity, particularly of the vessels near the macula, beaded appearance—accentuation of the central light stream and sheathings (whitish stripes bordering the vessels) of the sheaths, indentation of and lack of transparency at vessel crossings, locomotion pulse, changes in caliber, retinal infiltrations, in addition, edema (grayish opacity) of the retina, retinal hemorrhages, the pulsation pressure test phenomena, edema of the lids, recurring conjunctival chemosis, and subconjunctival hemorrhages.

### BRAIN ABSCESS

In December, 1930 a young man of twenty-four came under my care suffering from severe, persistent headaches. Several years previously he had had a right simple mastoid operation. Eye examination was negative. Other findings, however, suggested a suspicion of brain abscess, which was concurred in by an eminent neurologist. Blood examination, however, disclosed the *Plasmodium malariae* of Laverin and the diagnosis of malaria.

This case is cited more particularly to stress the value of "routine" in examination.

The diagnosis of brain abscess is almost always difficult. It is generally accepted that they have their origin as complications of infective processes originating outside the cranial cavity, such as diseases of the ear, mastoid, nasal accessory sinuses, heart, lungs, etc. In making the diagnosis of brain abscess, therefore, it is necessary to establish the causal rela-

tion between the original focus and in addition exclude as possible explanations of the symptoms presented a host of other diseases such as brain tumor, meningitis, apoplexy, cerebral syphilis, encephalitis lethargica, etc. A problem of this magnitude demands that every source of possible aid be utilized. The eye not infrequently exhibits symptoms in brain abscess which may, in connection with the other symptoms, prove of considerable value in the diagnosis and localization. An enumeration of the eye symptoms which may occur in the various types of brain abscess includes the following: Photophobia, diplopia, optic neuritis, papillitis, choked disk, nystagmus, pupillary disturbance, hemianopsia, oculomotor and abducent nerve paralysis and sensory aphasia. The most important of these is optic neuritis. According to Gowers, optic neuritis occurs in two thirds of the cases and if the abscess has existed for more than four weeks it is found in three fourths of the cases, also, that if it is limited to, or more pronounced on one side the abscess is likely to be on that side. Practically all observers agree that the optic neuritis, in particular the swelling of the papilla, is seldom as marked as in brain tumor, and also that optic neuritis and choked disk are more frequent manifestations of brain tumor and meningitis than of brain abscess. It is also said that optic neuritis and choked disk are more common with cerebellar than with cerebral abscess. Choked disk is generally regarded as an expression of extreme increased intracranial pressure, as may occur in brain tumor. It is not a usual manifestation of brain abscess but is more common in brain tumor, occurring in 71 per cent of cases, in brain abscess in 22 per cent (Uhthoff), due perhaps to the usual absence of increased intracranial pressure in brain abscess and its usual presence in brain tumor. Sharpe, for example, states, "Unlike most brain tumors (excluding the gliomas) which produce an increase of the intracranial pressure by their added tissue formation or by a blockage of the ventricles, brain abscesses, on the contrary, replace brain tissue so that unless the escape of cerebrospinal fluid from ventricles is blocked by a large subtentorial abscess for

mation there are produced no signs of a marked increase of the intracranial pressure”

Eggleton's deductions are, “A moderate grade of papilledema is a frequent manifestation of cerebellar abscess, papilledema is frequently seen in occipital lobe abscess, papilledema is usually absent in front lobe abscess, transient or fixed hemianopsia of the homonymous type, for form and colors is a valuable localizing sign of temporosphenoidal lobe abscess and should always be sought for, vertical nystagmus and lateral deviation of the eyes when present are definite indications of cerebellar involvement, ocular paralysis is of no localizing value”

Photophobia may be present in the early stages, but it is of no determining value as to localization or stage of abscess. Diplopia occurs occasionally in temporosphenoidal and frontal lobe abscess. Wyllie states that the state of the pupil does not assist in the diagnosing of the abscess but is of decided value in deciding the stage and the side on which the abscess is located—equal pupils which respond actively to light and accommodation indicate the early purulent stage, if the pupils lose their activity and become sluggish or stable it is an indication of the terminal stage, if unequal, one being widely dilated, the abscess will likely be found on the side of the widely dilated one, if both pupils are widely dilated the abscess is probably large. Vision may be affected and its prognosis is uncertain depending on all the factors entering into the case. While the eye symptoms of brain abscess are of aid in diagnosis their absence is not to be interpreted as an indication that a brain abscess is not present.

**Practical Eye Diagnostic Summary**—In all suspected cases of brain abscess, a field of vision for both form and color should be taken with especial emphasis on disclosing if an homonymous hemianopsia be present which is a valuable eye symptom, pointing to a temporosphenoidal lobe abscess. The findings on first examination may not be complete but suggestive, later, tests may confirm the early suggestive features, hence, all the foregoing eye tests should be repeated at frequent intervals. The pupils should be carefully examined for equality and reaction. The eyegrounds, especially the papilla, carefully studied for early evidences of optic neuritis.

## BRAIN TUMOR

In June 1930 a young girl of nineteen was referred to me for an eye examination to determine if glasses might not relieve the severe headaches from which she had suffered for some time. She had recently been operated for gallbladder infection the diagnosis of this condition being largely based on the presence of gastro-intestinal disturbance persistent severe headaches and jaundice. No relief resulted from the operation. Eye strain was considered as a possible cause of the headaches. The eye examination disclosed a well marked choked disk involving both eyes and the diagnosis of brain tumor was made.

The eye symptoms of brain tumor are of outstanding significance, diagnostically and prognostically, and frequently are determining factors in appraising the clinical progress of the cerebral lesion and its proper therapeutic management. It should be recalled, however, that these eye symptoms are not of themselves as a rule determining elements but must be considered only as a part of the clinical ensemble of symptoms presented. The chief eye symptoms of brain tumor are (1) Those involving the optic papilla and retina, (2) changes in the visual fields for form and color and alterations of the blind spot, (3) impairment of the light sense. Of these symptoms the most important is that affecting the optic nerve head, commonly referred to as choked disk, papilledema, papillitis, optic neuritis or by the German designation, "Stauungspapille." It is present in 80 to 90 per cent of cases. Foster states that choked disk is absent in only 5 to 10 per cent of cases and these are mainly tumors of the frontal brain and of the hypophysis. As a preliminary to the discussion of the symptom, choked disk, it is well to refer briefly to certain anatomical relations of the cranial cavity, brain, optic nerve and retina which have an important bearing on the evolution of this eye symptom and the general accepted theory of its production (Fig 152)

The eyeball, optic nerve and retina are essentially outgrowths of the brain. The retina is in reality an expansion of the brain and the optic nerve may be regarded as a cerebral tract. Each optic nerve trunk is made up of nerve fibers separated by neuroglia and held together by connective tissue

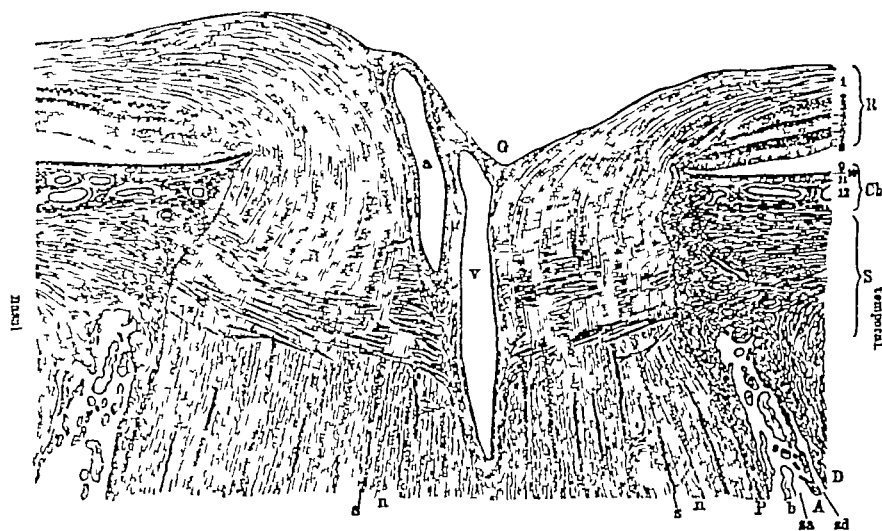


Fig 152—Longitudinal section through the head of the optic nerve. In its passage through the scleroticocochochoidal canal the optic nerve shows an irregular conical contraction. The fibers of the nerve are collected into bundles, separated by septa, *s*. Under the form of rows of nuclei, which belong to the neuroglia cells, the continuation of the septa can be followed to the head of the optic nerve. The axis of the nerve is occupied by the central vein, *v*, and the central artery, *a*, which is situated to the nasal side of the vein. The optic nerve is traversed transversely by the lamina cribrosa, which separates the trunk from the head of the nerve. The fibers of the lamina cribrosa arise from the wall of the scleral canal, traverse the nerve in a slightly concave arch (the concavity being directed to the front), and are inserted into the connective tissue that accompanies the central vessels. About at the level of the inner layers of the choroid, the nerve fibers diverge like a sheaf, so as to form a funnel-shaped depression—the vascular funnel, *G*. More fibers pass to the nasal than to the temporal side of the papilla, for which reason the former side is the higher. The fibers of the optic nerve pass over into the fiber layer (1) of the retina. Succeeding this are the other layers of the retina, namely the layer of ganglion cells (2), the inner plexiform layer (3), the layer of inner granules or bipolar cells (4), the outer plexiform layer (5), the layer of the outer granules or of the bodies of the visual cells (6), the limitans externa (7), and the layer of rods and cones (8). The layers of the retina stop short at the head of the optic nerve, the outermost layer, 8, extending the furthest in. The innermost fibers of the sclera which form the wall of the scleral canal, accompany the optic nerve backward and form its pial sheath, *P*, which is in intimate relation with it. At a point further back from the nerve head the outer layers of the sclera are reflected backward and form the dural sheath, *D*, which envelops the nerve loosely. Between these two sheaths lies a third, the arachnoid sheath, *A*, which divides the intervaginal space of the optic nerve into the subdural space, *sd*, and the subarachnoid space,

Lymph channels occur between the connective tissue and the bundles. The nerve trunk is surrounded by three enveloping sheaths, the dura, arachnoid and pia, which are the direct continuation of the cerebral meninges. Between the dura and pia is the intravaginal space which is divided by the arachnoid sheath into the lymph spaces, the subdural and subarachnoid. Both of these lymph spaces originate in and are continuous with the corresponding cerebral channels. These enveloping sheaths terminate by a cul-de sac in the sclerotic coat of the eye near the lamina cribrosa. The central vessels (artery and vein) of the retina run in the axis of the optic nerve to the optic papilla where they enter the eyeball and spread out over the nerve head as the central retinal vessels. They are presumed to have no anastomosis and are regarded as end arteries. The connection of the cerebral lymph spaces with the sheaths enveloping the optic nerve, and in particular their termination in a cul-de sac in the immediate neighborhood of the optic nerve, favors blockade and compression of the nerve at this point. This anatomical arrangement explains the readiness with which an increase of intracranial pressure may by compression affect the nerve head and retinal vessels and produce in the eyegrounds the familiar ophthalmoscopic picture of

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sa Anteriorly both end by a cul-de-sac in the substance of the sclera. *b* is the cross section of one of the numerous subarachnoid trabeculae which connect the arachnoid to the pial sheath. In the wall of the scleral canal is seen the cross section of some blood vessels belonging to Zinn's scleral circle. Between the sclera, *S* and the retina *R* lies the choroid *CA*. The innermost layer of the latter the lamina vitrea 10 is the one that extends the furthest in toward the nerve head and the fibers of the nerve are constricted by the edge of the lamina. Upon the lamina vitrea lies the pigment epithelium 9 which belongs to the retina and which on the nasal side extends as far as the lamina vitrea, but on the temporal side stops somewhat short of it. On both sides the pigment epithelium gets to be thicker and more pigmented toward its edge—a state of things which answers to the choroidal ring that can be seen with the ophthalmoscope. The succeeding layers of the choroid the chorio-capillaris, 11 and the layer of medium and large-sized vessels 12 do not extend quite up to the optic nerve on the temporal side because a layer of connective tissue representing a continuation of the sclera juts in between the two. (Fuchs-Duane Textbook of Ophthalmology " Courtesy of J. B. Lippincott Co.)



choked disk The fibers making up the optic nerve along with the vessels imbedded in their substance enter the back part of the eyeball through a rather intricate aperture known as the foramen sclerae and on reaching the interior eye spread out over its posterior inner surface to form a nerve carpet, the retina The optic nerve trunk does not enter the eyeball at the foramen sclerae through a single large opening but in the following manner (modified from Fuchs) The sclera divides into an outer and inner portion The outer portion, constituting two thirds of its thickness, is not perforated at all, but is reflected backward and enfolds the trunk of the nerve forming the dural sheath, the inner portion, along with a small portion of choroid process traverses inward forming a septum or diaphragm which is perforated by numerous openings through which the separate bundles of nerve fibers pass This perforated diaphragm resembles a sieve, hence the name "lamina cribrosa" applied to it The optic nerve at the foramen sclerae and especially at the lamina cribrosa is tightly enclosed between firm fibrous walls and when swelling of the optic nerve takes place, constriction and strangulation of it usually occurs Viewed with the ophthalmoscope, the lamina cribrosa, when present, appears on the disk surface as a small stippled zone made up of grayish dots and white interspaces The dots are the nerve fibers and the white interspaces are the septal partitions between the apertures The central vessels enter with the nerve fibers, divide and traverse the retinal surface The separation or diverging of the nerve fibers and vessels on the nerve head produces a funnel-like depression called the vascular or physiologic cup the bottom of which may present a stippled appearance, the lamina cribrosa The area of entrance of the nerve fibers and vessels to the eyeball is known variously as the nerve head, the optic disk or papilla The two optic nerve trunks proceed backward from the eyeball and join at the chiasm In the chiasm the fibers of each trunk divide, semidecussate and form optic tracts, each of which is a composite of nerve fibers from its own and the opposite side, a left and right The optic tracts

proceed backward respectively to the right and left primary subcortical optic centers and visual areas in the occipital lobe (Fig 153) The nerve fibers of accommodation, convergence and of the pupil pass from the optic tract to the oculomotor nucleus and from this nucleus send fibers in the trunk of the oculomotor nerve to the pupil, to the muscles of accommodation and convergence This semidecussation of nerve fibers at the chiasm and the distribution of the tracts explains the various pupillary, accommodation, convergence and hemianopic phenomena frequently exhibited in cerebral neoplasms and other intracranial disturbances which often provide important eye symptomatology in the diagnosis and localization of the lesion The chiasm rests upon the sphenoid bone and is in intimate relation with the pituitary body, the anterior end of the third ventricle, the infundibulum, internal carotids and the meninges

Many theories have been advanced to explain the development of choked disk, notably the mechanical and the toxic or inflammatory (Fig 154) The consensus of opinion favors the mechanical theory Cushing observes, "It seems in all probability a stasis edema from the forcing of the cerebrospinal fluid into the meningeal sheath which invests the optic nerve" Fuchs' comprehensive explanation of the way choked disk may be produced by an intracranial growth is of interest, "A brain tumor as a result of its growth arrogates constantly more and more space to itself within the cranial cavity Hence, as the skull is unyielding there arises an increase in the intracranial pressure Owing to this increase the return flow of lymph in and about the optic nerve is impeded and a stasis of lymph occurs producing an edema of the nerve trunk This edema causes a compression of the central vessels, a compression which makes its influence felt, sooner and to a higher degree, in the central vein of the optic nerve than in the central artery As there is constantly pouring into the papilla through the artery a quantity of blood which cannot be completely carried away again by the contracted central vein, venous engorgement of the optic nerve and consequently



swelling of the latter developed. This swelling of the nerve leads to its incarceration at the spot where it fits so tightly

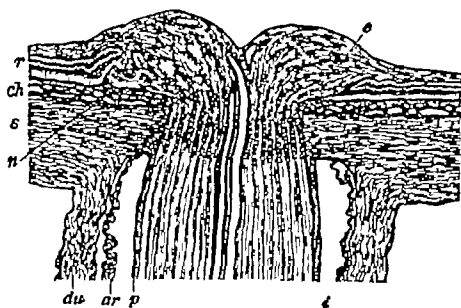


Fig. 154.—Longitudinal section through the head of the optic nerve in choked disk (magnified  $14 \times 2$ ). The disk is greatly swollen so as to project above the level of the adjacent retina and form at the base an annular protrusion, *n*. There is a cellular infiltration particularly along the minuter blood vessels, *c*, for which reason the latter appear specially prominent. The retina, *r*, is thrown into folds about the circumference of the disk in consequence of the swelling of the latter; the choroid *ch* and the sclera *s* are normal as is the optic nerve posterior to the lamina cribrosa. Here there is present, simply, a dilatation of the intervaginal space *i* through accumulation of fluid by virtue of which the greatly folded arachnoid sheath *ar* becomes especially prominent. *du*, dural sheath; *p*, pial sheath. (Fuchs-Duane Text book of Ophthalmology." J. B. Lippincott Co.)

in the foramen sclerae and consequently extreme edema develops in the strangulated nerve head. This edema constitutes what is known as choked disk which therefore consti-

radiation *S* the pupillary fibers, *m* go to both oculomotor nuclei *K* and *A*. Each of these latter consists of a series of partial nuclei one of which sends fibers, *P*, to the sphincter iridis, another sends fibers, *A* to the ciliary muscle (muscle of accommodation) and a third sends fibers *C* to the converging muscle (internal rectus) *i*. All three fibers run in the trunk of the oculomotor nerve, *Oc*. Division of the optic tract at *gg* or *cc* produces right hemianopsia and in the former case there would theoretically be no reaction to light on illuminating the left half of either retina (hemikinesis). Division of the chiasm at *ss* produces temporal hemianopsia. Division of the fibers *m* abolishes the reaction of the pupil to light but leaves the associated reaction of the pupil to accommodation and convergence unaffected. (Fuchs Duane Textbook of Ophthalmology. Courtesy of J. B. Lippincott Co.)

tutes a very important symptom of increase of the cerebral pressure" The ophthalmoscopic picture of choked disk has been summarized by Marcus Gunn in five stages

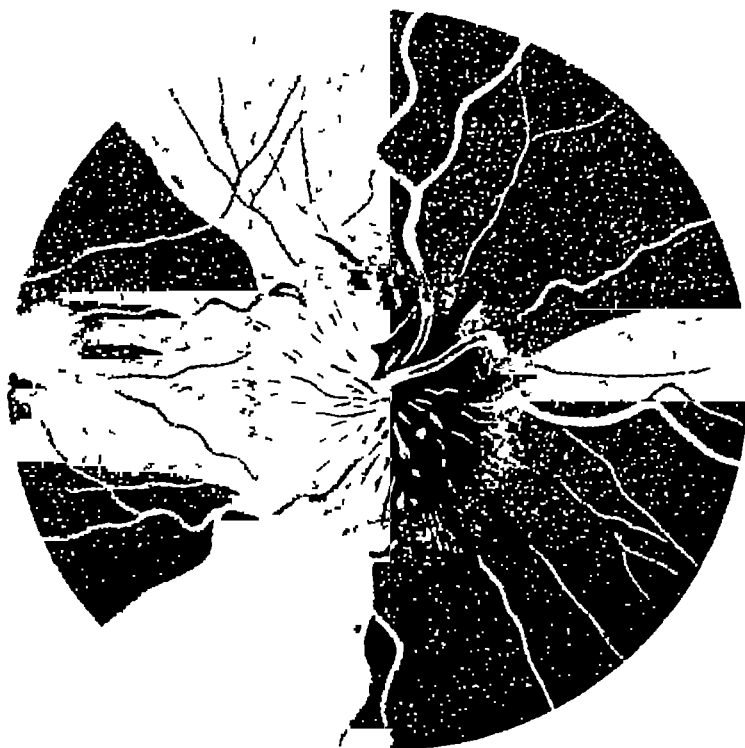


Fig 154a—Choked disk at its acme Scarcely a trace can be seen of the margins of the papilla, but the latter seem to send tongue-like processes into the retina The papilla is distinctly elevated, as can be seen from the course of the vessels, and exhibits a radiating striation A number of hemorrhages, also striate in form, give the disk quite a specific appearance The disproportion between the arteries and the veins is so great at the acme that the former are scarcely visible, while the latter leave the papilla as broad, tortuous bands Some white patches of degeneration are visible in the retina There are only a few retinal hemorrhages in this case, but they are often much more numerous The vision in this patient was normal (Adam Foster, "Ophthalmoscopic Diagnosis")

1 The earliest ophthalmoscopic signs are increased redness of the disk, loss of definition in its edges, slight prominence of its surface and narrowing of the physiologic pit

2 As a rate which varies much in different cases and which seems to bear a decided relation to the degree of intracranial tension, the swelling of the papilla increases, the physiological pit disappears and the disk edges become quite obscured. Along with these signs there is now slight haziness of the surrounding retina and the retinal veins show evidence of retarded circulation.

3 In an advancing case the next alteration consists in further swelling of the papilla so that it becomes more prominent and occupies a larger fundus area, the venous distention becoming more marked. Fine folds not infrequently appear in the edematous retina, particularly between the disk and macula and there may be retinal hemorrhages.

4 The papilla becomes more opaque and sometimes more prominent, the hemorrhages increase in size and number and there are inflammatory exudations on the disk and surrounding retina. At this stage vision has become impaired.

5 The next stage consists in a gradually decreasing vascularity of the papilla, part of its surface becoming paler than normal, while the prominence either persists or slowly subsides. At this time also we first note a change in the branches of the central artery in the form of diminished breadth—the state of atrophy with inevitable blindness.

From the foregoing summary it is evident that in making an ophthalmoscopic inspection of the eyegrounds for suspected choked disk our study of the nerve head should concentrate on the following five cardinal features: (1) Its color and transparency, (2) definition of its margins or disk outline, (3) condition of level, (4) appearance of the physiologic pit, (5) condition of vessels, ratio in size of the retinal arteries and veins, evidences of retinal edema and retinal hemorrhages. The various stage above outlined in an advancing process show step by step progressive involvement of these five cardinal features. The striking eyeground change of brain tumor is that of the nerve head, choked disk. It is markedly swollen and projects into the vitreous. Its edges overhang and the whole appearance resembles a gray mushroom. The vessels

emerging from the vascular funnel ascend over this mushroom swelling and may disappear as they descend its sides. The disk outlines, while more or less disturbed are still defined.

The second striking change is in the retinal vessels, the veins are increased in size and the arteries contracted. The height of the swelling is estimated with the ophthalmoscope by comparing the refraction of the surrounding retina with the summit of the disk elevation, 3 diopters corresponding to 1 mm of elevation. A generally accepted rule is that the disk swelling must show an elevation of at least 2 diopters in order to be classified as a choked disk. Sharpe deplors the usual custom of relying wholly upon a disk elevation of 2 diopters as an indication of intracranial pressure signifying a brain tumor. He emphasizes that when this amount of swelling occurs in the disk it is a late manifestation of intracranial pressure and of brain tumor and that the patient's chance of operative relief, conservation of vision, etc., correspondingly suffer when reliance is placed on such a late development. He points out that the earlier evidences of disk and retinal vessel involvement which indicate intracranial pressure should be looked for and accepted as definite evidences of increased pressure intracranially. These early signs are slight dilatation of the retinal veins and a blurring of the nasal margin of the disk, no "measurable swelling" of the disk being exhibited. These early disk and vessel changes must be examined frequently to note their progress and they should, he advises, be corroborated as evidences of increased intracranial pressure by lumbar puncture and the spinal mercurial manometer.

Visual impairment is exceedingly variable. It is often normal even in a well marked choked disk. It is not affected early in brain tumor due to the fact that the choked disk in such cases antedates the visual impairment and choked disk is, as a rule, a relatively late manifestation of brain tumor. Impairment of vision, therefore, is not a dependable symptom as a sign that choked disk is not present. It is necessary in every case of suspected brain tumor, even when the vision

is normal, to examine the eyegrounds to determine if a choked disk be present. In brain tumor the disk involvement is almost always sooner or later bilateral. Horsley believes that it is apt to appear earlier on the side of the lesion. Neither the size of the tumor, the age of the process, its situation, its unilateral exhibition or intensity are of dependable diagnostic or localizing value. Small tumors frequently cause choked disk while large ones fail to produce the symptom. Tumors of the posterior fossa (cerebellum) are the ones with which it is most frequently encountered and it is usually an earlier symptom in tumors of this locality than in tumors of the cerebrum. Cerebellar tumors and those in the midbrain and the thalamus are reputed to produce a more intense choked disk with rapid invasion of visual function than those of the cerebrum, the subcortical, parietal or frontal regions. Tumors of the parieto occipital regions, of the corpora quadrigemina and of the cerebellum are said to produce the highest percentage of choked disk and that pontine tumors and those of subcortical origin comprise the majority of cases in which the symptom is absent. The types of brain tumor usually encountered are glioma and sarcoma, in children the solitary tubercle. Choked disk is not exclusively a symptom of brain tumor, it may be present in albuminuric retinitis, brain injuries, concussion, apoplexy, thrombosis, the internal type of hydrocephalus, meningitis, etc. The visual field changes which may be exhibited are concentric contractions, possibly inversion of the color fields and enlargement of the blind spot. The pupillary symptoms manifested are occasionally of helpful diagnostic value. Radiations of white lines or dots about the macula resembling the "stellate figure" associated with albuminuric retinitis, sometimes occur (in 15 per cent, Patton).

**Practical Eye Diagnostic Summary**—There are no external changes of the eyeball which suggest a brain tumor. Vision may be normal in a patient with brain tumor even when there is a well marked choked disk present. Choked disk is a dependable symptom of brain tumor, occurring in 80 to 90 per cent of cases. The only safe course in all suspected cases particularly those which exhibit the familiar symptom triad of headache vomiting and vertigo



commonly associated with brain tumor, is to examine the eyegrounds with the ophthalmoscope. Such examinations should be repeated at frequent intervals. Choked disk is a relatively late manifestation of brain tumor. Early eyeground evidences of increased intracranial pressure are slight dilatation of the retinal veins and blurring of the nasal margin of the disk without a measurable swelling of the disk. These should be looked for and confirmed by lumbar puncture and the spinal manometer. The specific ophthalmoscopic evidences of choked disk are a grayish swelling of the disk surface of a mushroom character reaching a height of not less than 2 diopters, the definition of its margins being more or less maintained, the vessels being interrupted or disappearing as they descend along the sides of the disk swelling, the retinal veins being dilated, the arteries contracted. Such changes are, as a rule, bilateral but may be unilateral or more accentuated on one side. Any change, however, of the disk, such as increased redness, blurring of its edges, slight prominence of its surfaces, etc., should cause concern and the possibility of a brain tumor may be considered until it has been definitely excluded. A visual field test for form and color and of the blind spot should be included in the examination.

### EPIDEMIC ENCEPHALITIS

(Encephalitis Lethargica, Sleeping Sickness, Nona)

This disease has an added interest in view of the recent epidemics in Paris, Illinois, in 1932, and in St. Louis city and county in 1933.

All medical writers are in agreement that eye symptoms are almost always a conspicuous feature of this disease, particularly in its early stages, and that their presence in connection with other symptoms is of distinct value in establishing the diagnosis.

Barker, for example, points out that "In the acute or florid stage or first main stage, we meet most often with either, (1) a somnolent-*ophthalmoplegic* syndrome or, (2) an irritative hyperkinetic syndrome (either choreatic or myoclonic)," and he adds that "a triad of symptoms—fever, somnolence and *ophthalmoplegia*—characterize the lethargic type of encephalitis."

Among the eye symptoms which have been observed in this disease are the following: Ptosis, diplopia, accommodative pareses or paralyzes, *ophthalmoplegia* of all varieties, complete partial ex- or intrinsic, impairment of globe movement upward or downward, nystagmus, "oculogyric crisis," blepharospasm, inequalities and irregularities of the pupil, im-

paired pupillary reflexes, mydriasis, myosis, Argyll Robertson pupil, optic neuritis, papilledema, oscillation of the globe, photophobia, "myostatic rigidity" of the eyes, etc

An analysis of these ocular manifestations discloses that practically all of those enumerated have as their underlying pathology a paresis or paralysis of the nerves which supply eye structures and upon which these structures depend for functional integrity

The pareses or paralyses of the various ocular structures are more or less erratic and capricious in their exhibition, ranging from a paralysis of *all* the eye muscles, so-called total *ophthalmoplegia*, or attacking only all the external muscles of the eye, so-called *external ophthalmoplegia*, or perhaps only the sphincter pupillae and ciliary muscle, so-called *internal ophthalmoplegia*, or only paired ocular muscles, such as the external rectus of one eye and the internal rectus of the other, so-called *associated* or *conjugate paralysis*. Two outstanding characteristics are usually conspicuous in the eye symptoms of epidemic encephalitis (1) They are often temporary and evanescent in duration, erratic in course and behavior and exhibit unpredictable and unstable clinical varieties as to onset, tenure, recurrence or permanency, (2) frequently they also manifest a "selective" tendency, for example, of a group of ocular muscles supplied by the same nerve, only a few "selected" muscles—perhaps only one—of the group will be attacked. Various similar pareses and paralyses also occur in syphilis, tabes, diphtheria, influenza, rheumatism exposure to cold, various spinal and cerebral lesions, tuberculous meningitis, diabetes, toxic agents, ptomaine poisoning, etc., and these, therefore, must be excluded as etiologic factors. The presence of an Argyll Robertson pupil, for example, may be confusing. This pupillary phenomenon occurs quite regularly in tabes and syphilitic infection. Adler points out that if it be manifested in epidemic encephalitis it disappears rather promptly, while in syphilis it persists.

Pupillary disturbances, such as impaired reactions, in equalities, miosis, mydriasis etc., occurring in epidemic en

cephalitis are, as a rule, evanescent, vacillating, frequently disappearing quite promptly and perhaps recurring. In contrast, such phenomena occurring in spinal and other cerebral lesions are more stable, constant and persistent. The motor ocular nerves, notably the third and sixth, less commonly the fourth, usually bear the brunt of the ocular invasion and produce such symptoms as disturbance involving pupillary reaction, its form, size, etc., accommodative impairment, restriction of ocular and lid excursion, diplopia, ptosis, etc.

It is customary to refer to so-called early and late symptoms. Among the symptoms which are frequently referred to as early ocular manifestations are ophthalmoparesis, which Stevens states occurs in 75 per cent of the cases, the extrinsic muscles of the eye are affected more frequently than the internal. Moore states that of 168 cases reported by the London Government Board, in 20 per cent diplopia was an early manifestation, the symptom disappeared rapidly as the disease evolved. Late ocular symptoms, those associated with the so-called residual or of the postencephalic parkinsonian stage, are blepharospasm, nystagmus, convergence, paralysis, anisocoria, oculogyric crisis, myostatic rigidity of the eyes.

The oculogyric crisis symptom as referred to by Williamson-Noble, "consists of spasmodic attacks of conjugate deviation of the eyes, most frequently vertical and often associated with a marked emotional factor. It is apt to be regarded as a manifestation of hysteria." The myostatic rigidity of the eye symptom, so named by Cords but first referred to by Nonne, is described by Barlow as follows: "The face has a masked-like appearance, the action of the facial muscles appears to be entirely abolished, and there is not a trace of mimicry in the face, the eyes are expressionless and stare vacantly into space or at some fixed object for many minutes and even hours at a time. Stellwag's sign is also present. It is not a question of paralysis of the eye muscle in these cases, when the patient is aroused the eyes can be moved with an effort, but they soon assume the same fixed position." It is generally accepted that optic neuritis, papilledema or fundus

changes are infrequent manifestations Foster Moore believes that ptosis is the most common symptom, it is usually bilateral

**Practical Eye Diagnostic Summary.**—An eye investigation should be included in the general examination of suspected cases.

It should be particularly directed to the lid and globe movement, the appearance of the pupil and the pupillary reactions, the accommodative and convergence range and an inspection of the eyegrounds

### DIABETES

The ocular manifestations of diabetes are many and varied, involving the lens, optic nerve, choked disk, retrobulbar neuritis, retina, choroid, ciliary body, vitreous, external ocular muscles, the accommodation and refraction The incidence of ocular involvement in the disease is reported as from 20 to 33 per cent (Knapp) Groenouw classifies the eye lesions in order of frequency as follows Cataract 30 per cent, retinitis 23.5 per cent, optic neuritis 5.7 per cent, ocular muscle disturbance 3.9 per cent. Other manifestations occurring are refractive changes (including accommodation), vitreous opacities, iritis, thrombosis of retinal vein, hemorrhagic glaucoma and optic atrophy Of these ocular symptoms those most commonly met with in diabetes are *disturbances of vision accommodation and refraction, cataract, retinitis, and retinal hemorrhages* Hirschberg states that the most common ocular manifestation is a contraction of the range of accommodation DeSchweinitz believes that premature presbyopia with failure to accommodate is a common and early symptom Changes in refraction, an increase of hypermetropia or the development of myopia, also occur Inability to see clearly at the reading distance is common Disturbances of vision, particularly near vision, and the necessity for frequent changes of glasses is, therefore, suggestive of a diabetic ocular invasion Visual impairment is of frequent occurrence and in general is said to be more accentuated in diabetes than in albuminuria The degree of impairment is dependent upon the extent and the distribution of the fundus changes Fletcher states that "of the ocular complications cataract is the com

monest " Stevens observes "Cataract usually of the soft variety and bilateral occurs in about 5 per cent of cases, it may be present in diabetics young or old " Two types of cataract occur in diabetes, one in young patients which is characterized by a rapid onset and development, and the other in the old which evolves gradually Foster remarks that, "when a young person develops in both eyes within a few weeks from no known cause, cataracts which are evenly developed and when complete, may be seen by oblique illumination to be bluish white with sectors radiating from the center that have a luster like that of mother-of-pearl, an examination of his urine will almost certainly reveal that he has diabetes mellitus "

Fuchs' view is that "the prognosis of diabetic cataract, as far as the operation upon it is concerned, is less favorable than in senile cataracts, because in diabetes wounds show less resistance to infection and moreover diabetes predisposes to iritis " Most observers are in agreement with this view and urge that before operation be undertaken the diabetic process should be reduced to the minimum and the patient's physical condition improved to the utmost

**Diabetic Retinitis.**—Some difficulty may be experienced in properly evaluating the retinal changes due to an arteriosclerotic or albuminuric pathology which may coexist with the diabetes It is generally regarded that diabetic retinitis is not of frequent occurrence, that it is bilateral, that it affects mostly older subjects and that it is a late manifestation of diabetes DeSchweinitz mentions that in any case of diabetes of long duration, retinitis is seldom absent although it may sometimes be difficult to find the lesions because they may exist in the periphery of the eyeground and especially so if the complication of high myopia or cataractous lens is present Moore states that retinitis affects older people only and that it does not occur under the age of thirty-five Fuchs thus describes the retinal picture "It is characterized in many cases by the presence of small, brilliantly white spots in the retina which chiefly occupy the region of the macula lutea and

its vicinity, without, however, presenting a stellate arrangement as in albuminuric retinitis. Sometimes by the confluence of small dots one or two large patches are formed which show by their crenated borders that they are composed of smaller dots. Between the white patches lie punctate extravasations of blood. The rest of the retina is transparent and the papilla too is unaltered. In other cases this characteristic picture is not present, in fact, diabetic retinitis may actually appear

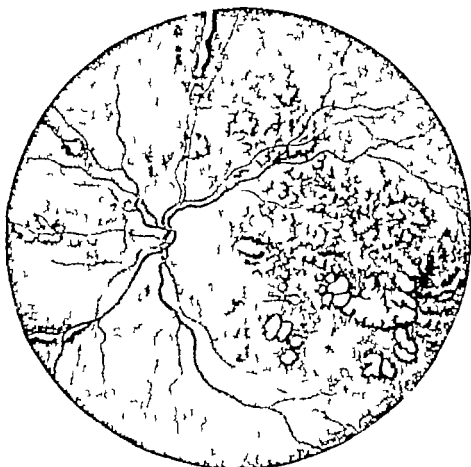


Fig. 1546.—Diabetic retinitis. Extensive white exudations in the macular region (deSchweinitz "Diseases of the Eye")

under the guise typical of albuminuric retinitis or closely resemble a retinitis circinata. Very rarely in young diabetics, especially when nearing death *retinal lipemia* is found, *i.e.*, a condition in which on account of fat in the blood the retinal vessels appear reddish white or pure white." Moore calls attention to some special features which he regards as particularly suggestive of a diabetic retinitis. (a) The patches of retinal exudate in diabetes tend to have sharp cut edges,

are often solid and soapy or waxy looking, are usually distributed in an irregular manner and sometimes form an irregular ring well wide of the macula, (*b*) a star figure is uncommon and if present it does not acquire the degree of symmetry that may be seen in renal cases, (*c*) the soft edged cotton wool patches so frequent in severe renal cases do not appear in diabetes, retinal edema is never so marked and thus retinal detachment does not result, (*d*) retinal hemorrhages are generally in the deeper retinal layers and, therefore, are roughly circular in outline instead of being flame shaped, (*e*) the circular retinal pigment spots which are not rare in the later stages of retinal retinitis are not seen in diabetes

Hirschberg describes a retinal picture called central punctate diabetic retinitis which he also regards as characteristic of diabetic retinitis. The optic disk is normal and the retinal vessels are not visibly sclerosed. There is no hyperemia or edematous clouding of the surrounding retina. In the zone adjacent to the optic nerve between the superior and inferior temporal blood vessels groups of punctate, small, discrete, sharply defined white spots are seen. Between the spots numerous small punctate or striated hemorrhages are found. The spots may extend to the nasal side. Thorington observes that "the snow bank about the disk and the macular star, commonly seen in albuminuric retinitis, are absent in diabetes."

**Hemorrhages**—Hirschberg (quoted by Knapp) divides retinal hemorrhages in diabetes into four groups (1) Small punctate hemorrhages, (2) larger hemorrhages with vitreous opacities, (3) hemorrhagic infarct of the retina, (4) hemorrhagic glaucoma. It is believed that diabetic subjects are prone to develop intraocular hemorrhages and that a diabetic retinitis exhibits more hemorrhages than the albuminuric retinitis. They are small and since their seat is below the nerve fiber layer their outline is more or less circular and not, as in albuminuric retinitis, flame shaped due to the location of the latter in the nerve fiber retinal layer. Small dotlike retinal hemorrhages point to a diabetic condition. The hemorrhages may be limited or extensive and vitreous opacities may result

from a leakage of blood into the vitreous. Rarely hemorrhagic glaucoma may similarly be set up. Iritis is uncommon. Retrobulbar neuritis may occur with a relative central scotoma. The prognosis for life in a well marked diabetic retinitis is to be regarded as unfavorable though somewhat better than that of an arteriosclerotic or albuminuric retinitis. The visual prognosis likewise is uncertain.

**Practical Eye Diagnostic Summary**—In all cases of suspected diabetes an examination of the eyegrounds should be made. The fundus changes are small brilliantly white spots in the macular area and retinal hemorrhages. The picture may resemble an albuminuric retinitis. Usually the retina and disk present no changes. Suggestive symptoms are disturbance of vision particularly near vision and the necessity for frequent change of glasses. Cataract is a frequent complication. The rapid development of a cataract in both eyes in young subjects is suggestive of the presence of diabetes mellitus.

### NEPHRITIS

In May 1933 a patient aged forty five consulted me for reading glasses. His general physical condition he regarded as excellent. He presented no external evidence of ocular difficulty. His vision was practically normal and his only ocular complaint was of slight difficulty in reading the telephone directory. Examination of the eyegrounds disclosed an albuminuric retinitis and on subsequent general examination the diagnosis of Bright's disease was made.

The value of eye symptoms in nephritis is unquestioned. Clinicians generally emphasize their importance in contributing to the diagnosis and more especially in predicting a prognosis. It is a common experience for ophthalmologists to discover first eye pathology, suggestive or perhaps determining nephritis in patients who had regarded themselves as being in good condition. These facts are so well recognized that an examination of a case of nephritis cannot be regarded as complete unless an eye investigation has been included. The list of eye symptoms which have been attributed to nephritis is an extensive one. The most important may be summarized as follows. Noninflammatory edema of the eyelids usually bilateral and involving, as a rule, the lower lids. Spontaneous subconjunctival hemorrhage, especially of the recurring type, occurring in elderly people, chemosis of the conjunctiva, epi-



scleritis, exophthalmos, diplopia, vitreous hemorrhages, albuminuric retinitis, retinal hemorrhages, retinal detachment, hyperemia of papilla, optic neuritis, papillitis, choked disk, optic atrophy, visual field changes, impairment of vision to blindness

Of these the most important is *albuminuric retinitis* which is said to occur in 33 per cent of cases It is exhibited most

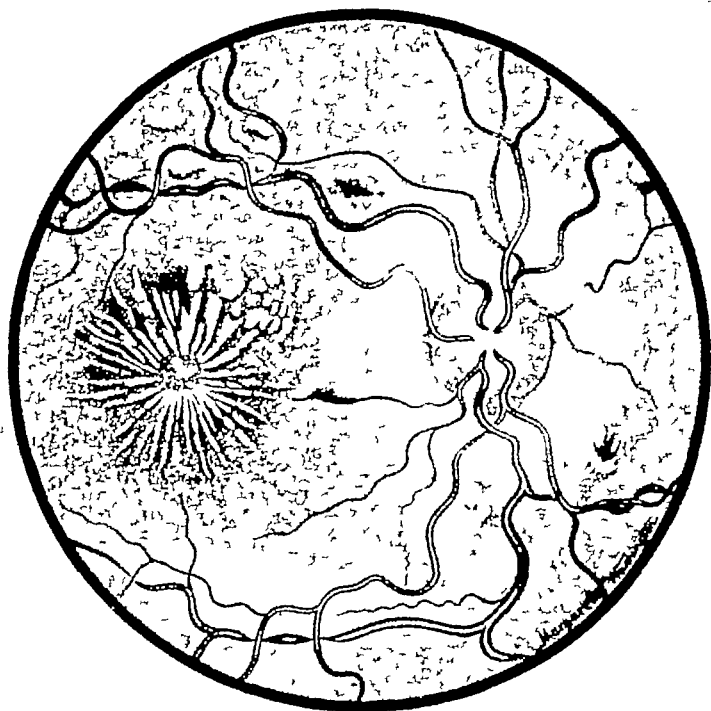


Fig 154c—Albuminuric retinitis, star-shaped figure in the macula, the circulation in the distended veins impeded where the latter are crossed by the arteries which are undergoing sclerotic changes (deSchweinitz, "Diseases of the Eye")

frequently in the small contracted kidney, next, in the chronic diffuse parenchymatous, next in the acute parenchymatous (scarlatina, etc ) and least in the amyloid degeneration It involves specially the optic nerve and papilla, the macular region, the retina and the retinal vessels The outstanding features of albuminuric retinitis of the typical type disclosed by

ophthalmoscopic examination are white spots in the region of the macula, these may take on the aspect of lines radiating from the fovea, but not necessarily invading it. Ultimately as a rule, these spots completely surround the macular region forming the so-called "star figure" The disk is encircled in toto or partially by round white or yellowish white masses which resemble snow Hence the name given them of "snow bank", retinal hemorrhages occur either minute or of greater extent, irregularly distributed and assuming round linear or flame-shaped forms The retinal vessels may or may not be obscured in areas by these and the vessels often exhibit changes characteristic of arteriosclerosis previously referred to The retina is swollen, edematous and in areas, as a result, where these changes are marked it is opaque. The optic papilla is commonly involved, varying in degree from hyperemia and neuritis to well marked choked disk Optic atrophy may eventuate as a sequel These various changes are associated with two pathologic forms, the *inflammatory* or *exudative*, or the *degenerative* Either of these types may be exhibited alone or in combination Depending upon the dominance of the several lesions the appearances, according to Gowers, may be designated as degenerative, hemorrhagic, inflammatory or neuritic The eye involvement is almost always bilateral Vision is affected, varying in degree from slight impairment to blindness, depending upon the severity, the stage, the character, location and extent of the fundus lesions Some degree of permanent impairment of vision is, as a rule, to be anticipated Retinal detachment may occur and is of serious import from the visual standpoint, the prognosis for reattachment being uncertain Occurring in the nephritis of pregnancy the prognosis is, however, more favorable The prognostic value of albuminuric retinitis is of outstanding importance There is a unanimity of opinion among clinicians that a well marked albuminuric retinitis in association with nephritis portends a tenure of life, in approximately 90 per cent of cases, not exceeding two years Some reservations as to the interpretation of the eye findings in albuminuric

retinitis are important to note. The ophthalmoscopic picture, for example, as Foster points out, is not determining in establishing either the variety of renal lesion or the stage of the disease. Neither is the "star figure" exclusively a sign, since it may be found also in retinal arteriosclerosis, diabetes, brain tumor, lead poisoning and acute infectious diseases, in changes affecting the optic nerve, the papilla and in choked disk, pernicious anemia and leukemia.

**Practical Eye Diagnostic Summary**—An examination of a case of nephritis without an inspection of the eyegrounds is incomplete. The eye findings, both diagnostically and prognostically, are too important to be ignored. The outstanding change to be looked for with the ophthalmoscope is the so-called "albuminuric retinitis," the characteristic changes of which are the star-shaped figure around the macula, the "snow banks" about the disk, retinal hemorrhages, edematous retina and involvement of the disk as an optic neuritis or possibly choked disk.

If time permitted many other general conditions which more or less regularly exhibit eye symptoms might be discussed in detail. Among these are the following

#### HEAD INJURIES AND SKULL FRACTURES

It is of the greatest importance in these cases to ascertain the state of the intracranial pressure. Such knowledge may safeguard the patient against compression of the medulla and be the deciding factor in operative decision. An increase of intracranial pressure is often manifested by an optic neuritis, choked disk or various pupillary symptoms. In all head injuries and skull fractures, therefore, an examination of the eyegrounds should be made and repeated at frequent intervals.

#### TABES

Eye symptoms are almost a regular accompaniment of tabes and are of definite value in establishing the diagnosis. The chief eye symptoms are Argyll Robertson pupil, which observers estimate occurs in 76 per cent of cases, often as an early symptom, optic atrophy in approximately 20 per cent of cases, also often an early symptom, ocular palsies and alterations in the visual fields, particularly those for color.

## SYPHILIS

All the structures of the eye, with the exception of the lens, may be affected by syphilis in any of its stages. The uveal tract—iris, ciliary body, choroid—are the structures most commonly involved. Both eyes are ultimately affected. A practical clinical observation is that in all eye diseases which prove intractable to treatment the possibility of syphilis being a factor should be excluded.

## PITUITARY BODY

Anatomically, the relation of the pituitary body to the optic chiasm is exceedingly intimate and in consequence eye symptoms are frequent and often a determining manifestation. Changes in the visual fields producing the various types of hemianopsia, are of special significance. Amblyopia, exophthalmos, pupillary phenomena, and nystagmus are also encountered. In a suspected case of pituitary disease a visual field should always be taken.

From the foregoing brief resumé of the subject, *The Value of Eye Symptoms in the Diagnosis of General Disease*, the following conclusions are warranted:

- 1 A very considerable number of general diseases exhibit eye symptoms during their clinical evolution and these eye symptoms are frequently of value as an aid to diagnosis and prognosis.

- 2 An eye investigation should be included as a routine measure in general physical examination. The details of such an examination are relatively neither excessively time consuming nor unduly technical and are definitely within the scope of the general physician.

- 3 Training in the use of the ophthalmoscope and the details of ophthalmologic examination in the study of general disease should be stressed and routinely applied in undergraduate teaching.



## CLINIC OF DR MICHAEL HIGGINS EBERT

### COOK COUNTY HOSPITAL

#### RINGWORM OF THE SCALP

Case I.—This boy of six is brought in by his mother because she has noticed "bald spots" in his scalp. The first of these appeared four weeks ago and now is as large as a silver dollar. The later ones are smaller. On questioning her we learned that a younger child in the family also has one or two similar spots. The disorder causes the child no discomfort. On examining the scalp we find that the involved areas are circular coin sized and not completely bald (Fig 155). There are a few stumps and stubs of

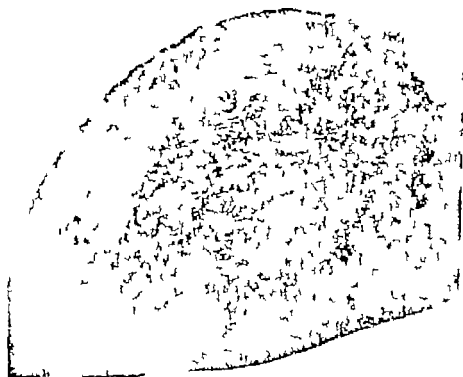


Fig 155—Ringworm of the scalp due to *Microsporon audouinii*

hair remaining which can easily be epilated. The whole area is covered with fine grayish scale. Had the mother used some home remedies, the scale might well have been absent. We pull out several hair stumps and place them on a glass slide, cover them with a coverslip and allow a few drops of 1 to 20 per cent potassium hydroxide solution to flow under the slip. The preparation is then set aside to be examined after an hour or two. A similar

preparation is made from the scales which are scraped off the scalp and placed on another slide. The potassium hydroxide solution softens the horny material in the hair and epidermal scales, and clears them so that fungus spores and mycelia become visible.

While the material is softening we will discuss the differential diagnosis of this case. Alopecia areata is not uncommon in children. It produces well defined circular areas of baldness which occur erratically in the scalp. If the scalp has not been treated, the baldness is complete in the affected areas when the lesion is recent. The surface is white, shiny, but not atrophic. If the process is active, short hairs, about  $\frac{1}{4}$  inch long, are found near the active margin which look like exclamation points. These hairs are nearly normal in caliber and color distally, but near the scalp are fine and atrophic. This gives them the appearance of an exclamation point. However, if the bald spot be of some duration, fine, new lanugo hairs may have regrown, giving it a fuzzy appearance. If the scalp has been treated with strong ointments or other preparations, a secondary dermatitis may result in the affected patches, making the diagnosis more difficult. The essential features are still present and there is no history of other members of the family being similarly affected.

Discoid lupus erythematosus may produce round bald patches in the scalp. These are dull red and may or may not be scaly. The follicular openings are dilated, and other similar plaques are usually found present in the ears or on the face of the patient. The patches heal with atrophic scars which result in a permanent loss of hair in the area. Lupus erythematosus in a child of this age would be unusual.

Now let us examine our preparations, first with the lower power of the compound microscope, cutting down the light to a minimum. We find that the proximal portion of the hair root is covered with a sleeve which is made up of a mosaic of thousands of tiny spores set closely together. Under the high dry magnification we find these spores look like little circles. The scales are next examined and are found to contain branching fungus mycelia. This is the characteristic

picture of a microsporon infection. The procedure I have described is not a difficult one. Anyone capable of ordinary work with the microscope can easily demonstrate ringworm fungi. Do not be in a hurry. Do not heat the preparations. Allow plenty of time to elapse before making the final examination. Examine several specimens. One should not make a diagnosis of ringworm of the scalp with its attendant social isolation without the microscopic demonstration of the fungus, any more than he would diagnose tuberculosis without demonstrating the bacillus.

The majority of microsporon causing lesions similar to the ones we have seen today are classified as *Microsporon audouinii*. Dr. Gruby, a Viennese who had migrated to Paris, first described this organism in 1844 and named it in honor of a famous French botanist. Exact species determination can be made only by an experienced mycologist by culture on various artificial media.

Occasionally we see cases of this type due to *microsporon lanosum*. This is a fungus of animal origin (cats, dogs, horses, etc.) It usually sets up a little more inflammatory reaction and is more susceptible to local treatment. This variety may attack the glabrous skin outside the scalp or even the beard region in adults.

Each one of these tiny spores has an extremely resistant membrane. Chemicals kill it with great difficulty. It is carried with the scales in the combings from the scalp. It adheres to the inside of the head dress and to the bed linen. If it finally arrives on another child's head, it germinates, sends out mycelia which penetrate the mouth of the hair follicle, grow around the hair root, penetrate the cuticle and finally produce a new crop of spores. You are all well aware that this is an extremely infectious disease. In institutions like orphan asylums, boarding schools, etc., it spreads like wildfire. It is so contagious that formerly special schools were set aside for its victims. It is a real problem even for the sporadic case, for he must be excluded from school just at an age when formal training is indispensable. It may even flare



up in great epidemics like the one in Basle, Switzerland, after the World War. Fortunately, the disease is self-limiting. Shortly after puberty, due to the action of the endocrine glands, a subtle change takes place in the hair and the sebaceous secretions which oil it, so that they are no longer a fit habitat for the *Microsporon audouinii*. Thus, a disease which was extremely difficult to eradicate now disappears spontaneously.

**Treatment**—Two factors conspire to render the treatment of ringworm of the scalp very difficult. First, the fungus proliferates deep in the hair follicles where it cannot be reached by ordinary measures. Second, the fungus spores are extremely resistant to parasitocides in a strength tolerated by the human skin. They can live long periods in dried material and still remain viable. The first difficulty is overcome by temporary epilation, the second by scrupulous cleanliness, keeping the scalp clipped short or better shaved, and by the use of antiseptics which will prevent germination of the spores.

The most effective agent for temporary epilation is the x-ray. This should not be immediately used in every case, however. Fortunately, practically all the forms of ringworm of the scalp with the exception of *Microsporon audouinii* respond to topical applications. If absolute species determination by a competent mycologist is not possible, local treatment should be given a thorough trial.

If local measures are to be attempted, any crusts which are present should be first softened by boric acid wet dressings and olive oil, and then removed. The hair should be clipped very short or shaved and washed thoroughly at least once a day with soap and water. The hair may then be epilated in a zone  $\frac{1}{2}$  inch wide about the affected patches with a tweezers or, better still, with a pencil made of pitch. An ointment is freshly prepared by adding 1 drachm of iodine crystals to 1 ounce of goose grease. This is rubbed well into the affected areas and surrounding zone every night. In the morning an ointment of 3 per cent salicylic acid and 5 per cent ammoniated mercury is applied to the entire scalp. The reac-

tion which occurs is a desirable one for it raises the tissue resistance to the fungus infection. However, we may have to abstain from the use of the iodine ointment when the reaction occurs and possibly use a boric acid wet dressing twice a day. The other milder ointment can usually be continued. As soon as the reaction subsides we resume the iodine ointment.

Whatever local application be used, frequent microscopic examinations of the scales and hair stumps must be made to check on the presence of the fungus. After all signs of fungus infection have been absent on repeated examination, the iodine ointment may be discontinued, but a 5 per cent ammoniated mercury ointment or 5 per cent sulphur ointment should be continued as well as the shaving and washing for two months, and repeated occasionally for short periods several months thereafter.

Darier, of Paris, recommends washing the scalp daily, drying it and then painting the whole scalp with a mixture of tincture of iodine 1 part and alcohol 3 parts, this painting to be continued daily or every other day depending on the amount of reaction.

Parkhurst, of Toledo, suggested a régime which has been found highly successful. He keeps the scalp closely shaven, washes it night and morning with soap and water, preferably tincture of green soap. He applies at night an ointment prepared freshly with 10 per cent fresh saturated alcoholic solution of iodine crystals in goose grease. In the morning he applies a salve made up as follows

Thymol	2 to 6 per cent
Ammoniated mercury	3 to 6 per cent
Salicylic acid	2 to 4 per cent
In benzoinated lard	

The strength of the active ingredients is gradually increased to the point of tolerance. This ointment is applied to the entire scalp.

During the treatment all the toilet articles should be kept separate and rigorously cleansed. The child should wear a

paper cap inside of his usual headdress and a similar one at night. These should be burned after a single wearing. Until the disease is well under control the child should not play with other children. Where there are several children in a family these should be frequently examined to detect incipient cases. Patience and scrupulous care are the most essential factors in the treatment.

If the infection proves intractable to these measures or if the hygienic environment is such that proper treatment of the patient and prophylaxis of other children is impracticable, arrangement should be made for x-ray epilation. Stimulating preparations should be discontinued for at least a week. The epilation should be done only by a physician who is adequately trained and has had plenty of experience. The amount of x-ray which produces a temporary alopecia is so near the amount which produces a permanent alopecia that one should no more attempt this procedure without special training than he would attempt a thyroidectomy without surgical apprenticeship. When properly performed, the hair begins to fall two weeks after the exposure and begins to regrow about one month later. The scalp should be washed carefully every day with soap and water until the fall is complete. Then it should be bathed daily with a 1 : 5000 solution of bichloride of mercury in 50 per cent alcohol, and a 5 per cent ammoniated mercury or sulphur ointment used daily for two weeks. Later this may be increased to a 10 per cent ointment. The scalp should be watched carefully for reinfection. The local treatment should be continued for several months.

The internal administration of thallium acetate will produce temporary alopecia of the scalp hair. The therapeutic dose is so close to the toxic dose that we are not warranted, I believe, in recommending this measure to the physician. The toxicity increases with the age of the child. In children near puberty the effective dose is almost always dangerous.

**Case II.**—Our next patient is a boy of nine, who presents what appears to be a large flat carbuncle on the scalp (Fig 156). This has been present for three weeks and is tender and somewhat painful. The lesion is well defined,

the size of a silver dollar raised well above the surface of the scalp. Its surface is dull red in color knobby and feels boggy. Through numerous openings a gelatinous material exudes. A few stumps of hair remain in the surface of the lesion but can easily be epilated. At first glance it will appear that if this lesion were laid wide open with a scalpel it would quickly drain and heal. However appearances in this instance are very deceptive. This boggy mass is due to another type of ringworm infection a trichophyton. The particular variety is of animal origin (ectothrix). Most ringworm infections of animal origin set up a considerable reaction in the affected skin. This reaction eventually kills the fungus so it is much harder to find the organism than it was in the first case where the *Microsporon audouinii* has learned to



Fig. 156—Kernion of the scalp

live almost in symbiosis with the body cells, producing a minimum of reaction and persisting a maximum of time. If we examine several of the stumps of hairs we may find a branching mycelium in the hair shafts.

Treatment should consist of hot wet dressings of boric acid or magnesium sulphate. Follicular pustules may have to be opened to promote drainage. The area should be painted daily with tincture of iodine 1 part, 50 per cent alcohol 3 parts. A preliminary epilation of the surrounding hair is advisable as in *microsporon* infections.

Nonspecific protein injections in the form of typhoid vaccine may be used in addition to raise the patient's resistance

A type of ringworm of the scalp rarely seen in Chicago but prominent in England and on the continent is the "black dot" or disseminated ringworm infection. It is easily missed unless the scalp is carefully examined for the lesions are small, inconspicuous and widely disseminated. They consist of scaling macules about a single hair follicle or a group of very few follicles. The affected hairs persist as stumps among their healthy neighbors. These stumps may turn on themselves, like a compressed corkscrew, beneath the scales or in the mouth of the follicle. Their appearance gives rise to the term "black dot." The majority of these cases is also due to a trichophyton but of the endothrix variety which is of human origin. Careful search of the stumps of hairs in a potassium hydroxide preparation will reveal the spores which are a little larger than those of *Microsporon audouinii* and are enclosed within the hair shaft, are quadrangular in shape and arranged in lines. All cases of localized dandruff should be suspected, especially if any of the hairs are broken off, and a careful microscopic examination made for the fungus.

Ringworm of the scalp may occasionally produce a generalized eruption of a toxic character which is termed an "id" eruption. When the original site of the infection is irritated or inflamed by treatment or secondary infection, some of the spores or perhaps their toxic products get into the general circulation and are carried to the peripheral cutaneous vessels. The skin has become during the course of the disease allergic to the products of the fungus and now reacts with an acute transitory inflammation which destroys any spores that may be present. So search for them is usually fruitless.

In kerion of the scalp a trichophytid is not uncommon. It is follicular, may be lichenoid, papular or consist of scaly patches. It is symmetrically disseminated on the trunk and lasts only a few days or a few weeks or may recur. It may easily resemble measles or scarlet fever. Microsporids are less frequent in occurrence.

The types of ringworm we have discussed, except kerion, heal without scarring. There is another fungus infection of the scalp, fortunately seldom seen in this country, which lasts a lifetime, gradually destroys the hair follicles, leaving an irregular atrophic scar beset with a few remaining wisps of hair. This is favus, caused by *Achorion schoenleini* (Fig 157). When well developed and untreated it produces small sulphur-yellow cups at the base of the involved hairs, giving off an unpleasant odor. It may involve the glabrous skin and the nails. The patients that we see here, however, usually come in with irregular areas of permanent baldness. It is



Fig 157.—Favus of the scalp

only by careful search of the scales taken from the margin of the atrophic areas that the fungus can be demonstrated. Favus is fairly common in southern and eastern Europe. Immigration officials have attempted to prevent its entrance into the United States. Nearly all the cases we see are immigrants with an occasional native-born patient from an infected family. The disease is contagious and intractable to treatment.

Since the patent medicine vendors have coined the priceless term "athlete's foot" and made America ringworm conscious via the radio, we constantly hear people say that they have athlete's foot of the hands or athlete's foot of the crotch.

Let us not commit the absurdity of "athlete's foot of the scalp"  
The fungus which affects the foot can never be transferred  
to the scalp So you can reassure unnecessarily disturbed  
parents who may confuse the terms and fear that they have  
infected innocent offspring with an organism originally picked  
up at a Turkish bath or golf club

## CLINIC OF DR SIDNEY A PORTIS

### COOK COUNTY HOSPITAL

#### GASTRO-INTESTINAL MANIFESTATIONS OF SYSTEMIC DISEASE AND THEIR DIFFERENTIAL DIAGNOSIS\*

THIS morning I would like to bring to you a group of cases that illustrate very clearly how one frequently might overlook some systemic disturbance because the patient complains of symptoms referable to the gastro intestinal tract. I think I have sufficiently emphasized to you in past lectures the more or less pathognomonic features and symptom complexes related to pathology *per se* in the abdomen. With this background it might be well for us to see what other disturbances could give these same symptoms and yet an exhaustive gastro intestinal examination give us little or no findings. I am sure you will agree with me that you cannot know gastro-enterology until you have learned general medicine. There are so many conditions outside of the abdomen that simulate intra abdominal disease that it is only the exceedingly myopic physician who sees only in the light of intra abdominal manifestations. Many patients are needlessly operated. Many patients are wrecked for their future life because some over-zealous physician has neglected to make a very painstaking and very thorough investigation of the patient as a whole. This not only includes a very careful detailed history with a searching physical examination, but also a very exhaustive laboratory and x-ray study. Before one can conclude that the pathology is in the gastro-intestinal tract and that it alone accounts for the symptoms, you must exclude beyond all rea-

\* Clinic given for students of Loyola University Medical School.



sonable doubt any extra-abdominal disturbance which might cause the patient distress

The first patient of this group of cases which I would like to present is a woman, forty-seven years of age, who consulted me on August 2, 1927 with a history in which her main complaints were belching, and a bitter taste in her mouth. The story is that she had a cholecystectomy and appendectomy nine months previously for the same complaints. No stones were found. For a short time following surgical intervention she was temporarily relieved, however, the same symptoms soon returned. She experienced fulness and distention particularly in the right upper quadrant, coming on at any time and lasting for a half hour, then disappearing spontaneously. She belches considerably and has noted a peculiar taste, not quite bitter, which as a rule comes on within a short time after meals. The distress is never present at night and disappears while resting. She complains of some aching across the back. Her appetite was normal. Her bowels were slightly constipated and she had occasional frequency of urination. The family history was of no importance. She had one pregnancy and no miscarriages. Menstrual history was regular up to the menopause at forty-four. Tonsils and adenoids had been removed at the age of forty, and the cholecystectomy and appendectomy done when she was forty-six.

Physical examination revealed a moderate enlargement of the thyroid gland. There was a coarse tremor of the hands and tongue. Reflexes were intact. Lungs were negative as was the heart. Blood pressure measured 102 systolic and 74 diastolic. There was some tenderness over the right rectus scar through which the gallbladder and appendix had been removed. There was some tenderness over the sigmoid region. The abdomen otherwise was essentially negative. Bimanual examination revealed moderate relaxation of the perineum, uterus anteфлекed and slightly enlarged, and some thickening in the region of the appendages.

Laboratory work revealed the urine containing an occasional pus cell but otherwise essentially negative. The stool showed some undigested food, a little mucus but otherwise was negative. There was no evidence of amebae or cysts. The stomach acidity was 10 free and total 30, with no evidence of obstruction. In the Ewald meal free acid was 35, total 45. Hemoglobin was 93, red count 5,000,000, and leukocytes 8200, with a normal differential count. Wassermann and Kahn tests were negative. The basal metabolism rate was — 14.

Fluoroscopic examination of the chest and abdomen revealed the lungs essentially negative as were the heart and aorta. The stomach was normal and the duodenal bulb filled out normally. The stomach emptied in four hours. Subsequent observations on the bowel revealed normal passage through the colon of the barium given by mouth.

In this particular patient with her gastro-intestinal manifestations, with subsequent surgery for them without improve-

ment, a very exhaustive and intensive study failed to reveal any definite cause for her disturbance. One might be prone in this case to think that her symptoms were entirely functional. However, clinically the patient did not impress me as one who would be predisposed to functional disturbance. She seemed like an exceedingly stable individual. There was one curious fact that might have been overlooked in her clinical story and which was reemphasized on going over the picture with her. That was that when she had a desire to urinate it must be satisfied within a relatively short time. She did not emphasize or mention it in her first visit when her history was taken, but in her story she alluded to a urinary disturbance which was not of much importance to her. Following this line of thought, it suggested itself that the patient might be cystoscoped to see whether any pathology might be found in the urinary tract to explain the symptoms. Interesting as it may be, examination of the urethra revealed a firm, fibrous stricture 2.5 cm long at the outlet. This was followed by a No. 24 F. Cystoscopic examination revealed a markedly trabeculated bladder, especially on the floor and posterior wall. Both ureteral orifices were normal and there was a moderate trigonal cystitis.

Curious as these findings may be, they are important as far as the patient is concerned because had they been recognized some time ago, useless surgery would have been prevented. Following adequate dilatation at repeated intervals, the patient's gastro-intestinal manifestations entirely disappeared. Her whole outlook on life was very much improved. She is now able to go about, doing her normal daily work, and has entirely forgotten that she has been ill. Yet, had not this patient's urinary tract been very carefully investigated she would have continued to go on from doctor to doctor, probably had more surgical intervention, and been no better off in the end. We were not justified in telling this particular patient that her urinary tract was cause of her symptoms until we had ruled out other causes in the intestinal tract which might account for her symptom complex. In a few

moments I will discuss with you why patients with genito-urinary disturbances give gastro-intestinal manifestations

The next patient I would like to present is a young married woman of twenty-seven who consulted me on August 20, 1923, complaining of pain in the right side, nausea, lumbar pain, headaches, loss of weight, and a tendency to perspire easily. For the past five years she had pain in the right side which was more or less constant. Appendectomy four years previously for this complaint failed to relieve her symptoms. The pain radiated up into the back and to the right scapula. She felt nauseated when the pain was at its height. It was never referred to the pubic region and she never required opiates for relief. There was some belching and distention after meals.

The noteworthy physical findings were a systolic murmur at the base of the heart, a blood pressure of 142 systolic and 90 diastolic. There was some tenderness over the gallbladder region, and a mobile right kidney was observed. Subsequent gastro-intestinal roentgenograms revealed a prepyloric spasm on the lesser curvature of the stomach which was distinctly tender. Otherwise, the gastro-intestinal examination was essentially negative.

She was treated for a period of three years off and on under gastro-intestinal management, with improvement and relapses. At this time, with the advent of cholecystography the gallbladder was investigated and it was thought to be pathologic. However, her symptoms in general overshadowed the findings in the abdomen. It was thought wise to investigate the urinary tract, because of this mobile right kidney. So, in 1928 Dr. Herman L. Kretschmer reported the following:

"Cystoscopic examination was negative except for a freely movable right kidney. The pyelogram showed the right kidney pelvis in normal position with the patient lying flat on the table, and a prolapse of the pelvis down to the crest of the ilium with the patient sitting up. The kidney could be easily displaced, so that the pelvis could be seen over the spine."

After much discussion and with some reluctance on the part of the urologist it was decided to do a right nephropexy. This was subsequently done and for the last seven years the patient has been completely relieved of all her symptoms and has had no recurrence at all of her gastro-intestinal manifestations.

Another similar case to this only in a male, was that of a patient, aged forty-seven, who consulted me in 1929 complaining of abdominal distress, belching and distention. For the past year or more the patient had had regular, recurring periods of abdominal distress coming on about an hour after meals, especially after the intake of fried and greasy foods, associated with fulness, distention and excessive belching. The past history other than a herniotomy fourteen years before and an appendectomy eleven years before was essentially negative.

Physical examination revealed no noteworthy manifestations. Examination of the urine and stool was negative. The gastric content was normal. Roentgenological examination of the gastro-intestinal tract revealed normal stomach and duodenum. Cholecystogram revealed what appeared to be a

pathologic gallbladder. Subsequent gastro intestinal management made little improvement in the patient's complaints.

During the course of observation a symptom of frequent and severe distress of the urinary tract developed. Cystoscopic examination was thought advisable. At this time there was revealed a residual bladder urine of 3 ounces and normal bladder tolerance. There were slight, coarse trabeculations on the posterior wall of the bladder, normal ureteral orifices, a depressed trigone with median lobe enlargement, which was definitely elevated and hypertrophied but with lateral lobes within the limits of normal.

Following transurethral resection of the prostate the gastro intestinal symptoms have completely disappeared.

Is it any wonder then that gastro enterologists might become urologically minded occasionally and search at times in the urinary tract for a possible explanation of the gastro-intestinal manifestations? I do not want to leave you with the feeling that this may be a panacea for all gastro intestinal disturbances, but there is a certain group of these patients which often will continue to be overlooked unless this possibility is sufficiently brought home to them either through their personal experience or by listening to the advice of those who have had trials and tribulations in cases of this sort.

Why should urological diseases give gastro-intestinal manifestations? E. C. Smith in an article some time ago very carefully showed that there was a very definite interrelation of the nerve supply of the upper urinary tract with the organs of digestion. He maintained that since both are supplied by the vagus and sympathetic nerves, any disturbance of the kidney may be reflected through the gastro-intestinal tract. The connecting link is through the celiac ganglion. Therefore, it is reasonable to suppose that impulses originating in the urinary tract can produce gastro-intestinal symptoms without pathologic changes.

The next patient I would like to present is a young lady twenty-seven years of age who came in because of pains in the lower abdomen "dyspepsia," and frequency of urination. Since 1933 the patient has complained of marked frequency and urgency of urination sometimes urinating as much as six to eight times at night. There was no evidence of any hematuria or pyuria. During this time she has complained of indigestion consisting of heaviness and a feeling of distention in the epigastrium occurring about fifteen minutes after meals, occasionally relieved by alkalis associated with much belching.

and heart burn, particularly after greasy foods. She had been somewhat constipated. There had been occasional nausea but no vomiting, and no hematemesis. She gained in weight. She is very irritable, has crying spells, some vertigo and faints frequently.

Physical examination other than slight tenderness over the region of the gallbladder and appendix is essentially negative. The other noteworthy findings are the absence of a corneal and pharyngeal reflex. Bimanual examination was negative.

The urine and stool were essentially negative. Stomach acids were 68 free and 80 total, with Ewald meal 90 free and 100 total. There was no evidence of retention. The blood showed evidence of a secondary anemia with a normal differential. The Wassermann and Kahn were negative. The basal metabolic rate was — 6.

Fluoroscopy of her chest revealed no unusual findings. Stomach was prolapsed into the pelvis for a distance of 2 inches, but was otherwise negative. The duodenum showed a spasm on the greater curvature side at its middle. The second portion showed evidence of stasis and surging. It was curled upon itself and had an S-shaped arrangement. The stomach emptied at the end of four hours.

Subsequent observations at twenty-four and forty-eight hours revealed a definitely tender cecum. The appendix was not visualized and the bowel was moderately spastic.

Cholecystogram showed a normal gallbladder as far as function was concerned.

Chest stereograms revealed no unusual pathology.

Urological examination revealed external genitalia negative, the urethra slightly narrowed, and to quote the urologist her history is entirely inconsistent with the clinical findings. "I do not believe the slight stricture of the urethra which she has is the cause of her frequent urination." To amplify this latter statement, dilatation of the urethra failed to reveal any of the symptoms.

On suitable gastro-intestinal management and with corset and pad to hold up the stomach, no improvement was noted. Following this a course of hospitalization was advised, with elevation of the foot of the bed and the usual measures adopted for the treatment of viscerotonic individual.

Still there was no improvement. The pains in the abdomen were migratory. It was then decided that possibly there might be some other cause or group of causes which might be accountable for her symptoms, but could not be demonstrated on the basis of organic findings. It is interesting to note that the girl was married at the age of nineteen, was married for three years and then separated for five years. She had three pregnancies, two interrupted and one child living and well. These periods of distress seemed to be definitely related to her sexual habits. Realizing that this might be a basis for her symptom complex, an analytic study was made. It is interesting to note that much of her gastro-intestinal symptoms are closely associated with her inhibited sexual desires. Under the guidance of a very competent analyst the patient has made steady and definite improvement and much of her gastro-intestinal symptoms have entirely disappeared.

This particular type of a patient is one who usually shows a battle-scarred abdomen and is frequently opened up for surgical intervention at the least provocation. I call it to your attention this morning because there is altogether too large a group of these patients who are going around from physician to physician and from surgeon to surgeon in an attempt to cure their gastro-intestinal complaints, when a careful, painstaking analysis of their symptom complex with the associated findings might lead one to believe that there is a so-called functional basis for their complaints. However, you are not justified in concluding that the functional basis alone is a causative factor until you have made an exhaustive laboratory and x-ray study to rule out the presence or absence of organic pathology.

The next patient is a married woman, forty years of age who came in because of a bowel distress. For several years she has noticed a tendency to loose stools. She has been told that she has colitis. She knows that if she avoids certain foods she is better. She has lost about 4 pounds in the last few months. She has also noticed some bleeding hemorrhoids. Her past history was that she had one brother and one sister die of pulmonary tuberculosis. She has had two children in the eleven years of married life both living and well. She has had the usual childhood diseases. About a year and a half ago had a so-called "influenza" with some hemoptysis and yet complains of no symptoms referable to her chest at the present time.

Physical examination showed some dullness on the right side of her chest, especially in the middle portion. There were numerous moist rales, "clicks." The heart was essentially negative. The liver was not palpable but the spleen extended 1 inch below the costal margin not tender. There was some tenderness along the course of the colon. Bimanual examination showed no unusual findings. Rectal examination showed large external and internal hemorrhoids. The urine showed no noteworthy findings. The stool was essentially negative except that acid fast bacilli were found. The stomach tests were within range of normal. Blood showed a secondary anemia of mild grade. Wassermann and Kahn were negative. Basal metabolism was -1 per cent. Her agglutination tests for bacillary dysentery organisms were negative as were the complement fixation tests for amebiasis. The sputum showed numerous acid fast bacilli.

Stereograms of her chest revealed evidence of involvement of the right upper and middle lobes. No evidence of cavitation could be seen. Fluoroscopic examination of her stomach and duodenum was negative.

In this particular case the patient was complaining of evidence of a so-called "colitis" for several years but more in the

last year. She had not lost any weight. There were no chest symptoms, yet one would be willing to believe that her gastro-intestinal symptoms were entirely related to her pulmonary tuberculosis. And since she has been under management at the sanatorium the more or less subacute manifestations of a tuberculous infection are subsiding, her gastro-intestinal symptoms are improving. No attention at all is paid to diet or to foods that formerly disturbed her so far as her so-called "colitis" is concerned.

Many patients with incipient, subacute, or even active tuberculosis show symptoms referable to the gastro-intestinal tract and it is only with a painstaking examination that these facts are brought to light, and in many cases pulmonary findings of this type are accountable for gastro-intestinal disturbance. When the pulmonary pathology becomes quiescent, symptoms referable to the abdomen entirely disappear.

Closely allied with this group of cases and very frequently mistaken in their diagnosis are a group of patients who have disturbances of the thyroid gland. Very frequently patients with symptoms of so-called "masked hyperthyroidism" present a gastro-intestinal picture which is so bizarre in nature and so prominent that the thyrotoxicosis is entirely in the background. This group of cases forms one of the very common sources of error in diagnosis of patients who consult me from time to time. Curious as it may be, it has not only been confined to young individuals but also is seen in elderly patients. Hyperirritability of the gastro-intestinal tract associated at times with hypermotility is a frequent cardinal symptom of this condition.

For instance, as we glance over the next patient, who is a married woman, fifty years of age, who comes in because of pain in the epigastrium and who states that for the last eleven years she has had dull pains in the epigastrium which had no relation to foods. She vomits frequently five to ten minutes after meals, which only slightly relieves her distress. Five and one-half years previously a cholecystectomy was performed for the same symptoms, with only transient relief. She has a great deal of nausea and some belching. Food taking only partially relieves her distress. There is no particular seasonal variation to her discomfort. Three years ago she had an appendectomy, but this gave her no relief. She has been gradually losing weight.

In her past history she had her menopause at thirty nine. She had six pregnancies, three children living and well. She occasionally got up at night to urinate. Her appetite was poor, due to distress when eating and there was some tendency to constipation.

Physical examination revealed a moderately obese woman. Thyroid was just barely palpable. The eye muscles only showed a slight weakness. There was a coarse tremor of hands and tongue. The reflexes were intact. The lungs were essentially negative. A systolic murmur was heard over base of heart. Blood pressure measures 150/70.

The abdomen showed numerous scars. There was a large hernia where the appendix had been removed.

Examination of urine and stool revealed nothing unusual. Her stomach acids were absent and 10 free in motor meal, with no evidence of retention and 20 free and 25 total in the Ewald meal.

Fluoroscopic of her chest and abdomen revealed a large shadow in the substernal region resembling a thyroid. The lungs were essentially negative. The heart was slightly enlarged in the transverse diameter. The stomach was negative. The duodenal bulb filled out normally.

Her Wassermann test was negative and her blood showed a slight secondary anemia. Her basal metabolism was + 62 per cent.

In this particular case no mention was made at all of nervousness, irritability and palpitation and all the symptoms that usually go along with ordinary thyrotoxicosis, and yet over a period of years this lady has had a low grade thyroid disturbance, with gastro-intestinal manifestation, subsequent surgical interference with no relief. We find on repeated metabolism tests the this is an out and out case of thyrotoxicosis.

Particularly interesting is it that she has a substernal thyroid. Following a subtotal thyroidectomy the patient was completely relieved of her gastro-intestinal symptoms, and for the last three or four years has been very comfortable.

If we look at the opposite picture it brings to mind a patient who is seventy-eight years of age who was complaining of gastro intestinal symptoms, accompanied by pain discomfort and obstinate constipation. She gives a history of the last fifty to sixty years of persistently taking cathartics with only partial relief. Very exhaustive examinations in the past had yielded no results. She was told by a doctor that she had a carcinoma of the stomach. However one observation of this patient, a very fat myxedematous type of individual who had apparently lost no weight would immediately give you the inference that possibly she was over a period of years a hypothyroid individual. Exhaustive examination of her gastro-intestinal tract revealed no evidence of malignancy and no cause for her long standing constipation. Her



basal metabolism was — 25, and nothing more was done for this patient than to put her on small doses of thyroid three times a day. No particular attention was paid to diet. The patient is now eighty-three years of age, still continues to take her small doses of thyroid extract and has a daily bowel movement.

Oh, how frequently are patients treated for years and years for constipation, if the clinician would only be alert to know that hyperthyroidism is a very common etiologic factor in producing the so-called "picture of constipation." You are not to believe that hypothyroidism is particularly confined to only obese individuals. It is frequently found in individuals who have normal weight or even weight below their standards. Both these disturbances of the thyroid gland give gastrointestinal manifestations and yet as far as organic pathology of the gastro-intestinal tract is concerned, it is entirely lacking.

Just the other day, there entered my ward service a patient, some fifty years of age, who had lost some weight and who had been treated for a period of months for gallbladder disease because he had associated nausea, vomiting, some belching, and some fulness after meals. The one symptom which the clinician did not pay enough attention to was that this patient had persistent headache, and furthermore associated with these headaches he had a slight auditory disturbance on the right side. While I am perfectly willing to admit that headaches may be a common symptom of gastro-intestinal disease, I would like to emphasize that the persistence of headaches after the presumed gastro-intestinal disturbance has been under control, should lead you to suspect the possibility of some other cause for the headaches. Two outstanding conditions come to mind, one the possibility of a cerebral neoplasm and the other, a so-called "indurative headache" associated with a hypertrophic osteo-arthritis of the cervical spine. In this particular patient subsequent neurological examination and ventriculograms revealed that the patient had a neoplasm at the cerebellopontile angle. The patient died before operative interference could be done. Necropsy examination revealed a tumor at the cerebellopontile angle.

This is not an uncommon source of error in gastro-intestinal diagnosis and it only goes to reemphasize to you the need of careful observations in each patient as to the cause of their particular complaints.

And how often is the clinician taken off his guard by such a patient as follows

A young woman twenty five years of age, who complained of headaches nausea and constipation. Patient said she first began to notice constipation ten months ago during pregnancy. Constipation continued through the period of gestation and was later relieved. She states that she had headaches which involved the entire head and were much more constantly present in exacerbations and remissions. These sometimes keep her awake at night, and they are so severe that she sometimes becomes nauseated and faints, and has blurred vision and black spots before her eyes. Of late she has been more or less persistently nauseated. Nausea has no relation to meals or types of foods. It lasts for about two hours and then disappears. She stated that she had had jaundice at five years of age. Her past history was essentially negative other than the jaundice as was her family history.

Physical examination revealed that she was quite tender over the gall bladder less so over the appendiceal region. Exhaustive studies of her gastrointestinal tract revealed no pathology to account for her symptoms, and in spite of management these attacks continued to recur. She developed some dizziness. This vertigo became a prominent symptom in her clinical picture. Exhaustive ophthalmological otological and neurological examinations by very competent consultants failed to enlighten us as to the possible source of her symptoms and complaints. And yet, in spite of it all symptoms persisted. With all these findings essentially negative there still must be some source for these manifestations and she was therefore told to see a psycho-analyst who found a very definite incompatibility at home associated with a fear of pregnancy. Following management in the hands of the psycho analyst her symptoms have entirely disappeared. The patient is exceedingly happy and is very comfortable.

A similar case to this is a patient who has had a diarrhea for years and years and years ever since she has been married. The most exhaustive search for the cause of the diarrhea from a gastro intestinal standpoint revealed nothing. There were no allergic manifestations to serve as etiologic factors. Feeling that there must be some reason for these gastro intestinal upsets she was also referred to a psycho-analyst. He found that there was incompatibility in the home and some brutality on the part of the husband in presumably intelligent surroundings. Since these have been corrected she is entirely free from her gastro-intestinal symptoms. This further emphasizes how frequently large bowel disturbances may be on the so-called "functional basis." This observation has been made so repeatedly that even the laity are acquainted with the influence of nervous disturbances on the so-called "stomach and bowel."

I have repeatedly called to your attention the numerous cases of cardiac involvement which come in complaining mainly of gastro-intestinal symptoms. When one realizes the marked hyperemia of the liver and the hyperemia of the gastro intestinal tract, is it any wonder that these patients complain so frequently of abdominal symptoms? These manifestations

may become so acute that to all intents and purposes they simulate an acute surgical abdomen, particularly resembling that of acute gallbladder. And to carry the clinical picture a little further, these patients may frequently develop jaundice and it has been explained by some that this bilirubinemia is associated with infarction in the lung, and the increased amount of serum bilirubin liberated is just enough to be retained in the blood stream due to the rise of the liver threshold for the secretion of bilirubin. So one should be careful in coming to too rapid a conclusion regarding a surgical abdomen in the presence of a decompensated heart. Coincident with the improvement of the cardiac manifestations, so do the gastrointestinal symptoms subside. Similarly, one sees gastro-intestinal manifestations in disturbances of the kidney.

Low grade nephritides or chronic uremia may have no symptoms at all pointing to the urinary tract. The entire clinical picture may resemble that of a disturbance in the abdomen. I do not emphasize at this time those acute manifestations of kidney pathology which resemble acute manifestations in the abdomen, but I am sure they have been sufficiently emphasized to you in your other courses. Here again under therapeutic management in those cases which respond to our best known methods of therapy at the present time in our attack on renal disease, the abdominal picture completely subsides. These manifestations do not resemble any particular disease of any one organ and it is for that reason they offer some difficulty in diagnosis from the abdominal viewpoint. How often does one see in women complaints which they think are entirely digestive in origin that may be associated with pathology in or about the pelvic organs. Just recently there was called to my attention a young woman who had been repeatedly treated for various diseases of the gastro-intestinal tract which on very careful search I found nothing which would account for her gastro-intestinal symptoms. But I did find a very large, incarcerated ovarian cyst which, when removed, entirely cleared up clinical manifesta-

tions Tubal and uterine pathology are equally responsible for upper abdominal manifestations

Now, while I admit that maybe I have left out some systemic manifestations which cause abdominal disturbance, I have tried to bring home to you this morning the one important outstanding clinical fact, that the clinician should never be gullible, the fact that a patient complains of his or her stomach is no reason at all for you not to read between the lines and find out if there is remotely some other cause for those symptoms When examining a patient for gastro intestinal symptoms, you begin at the head and go right down to the feet as you would in any other type of examination and then begin to correlate your abdominal findings with findings elsewhere Then you can make an exhaustive study from the laboratory and x ray standpoints to see if there is enough pathology in the gastro intestinal tract to account for the symptoms Finding that pathology lacking, it may be necessary for you to search further as to the possible cause of the disturbance It is only by this type of clinical practice you are able to arrive at a more accurate conclusion and a more accurate diagnosis in any one given case



## CLINIC OF DRS SOLOMON STROUSE AND HERBERT F BINSWANGER

MICHAEL REESE HOSPITAL

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### TREATMENT OF COMA

SINCE coma is a symptom of many underlying conditions it is axiomatic to say that the treatment of coma must depend on the nature of the etiologic factors involved. Therefore it would be unwise to discuss treatment without at least a cursory analysis of the causes. In the diagnostic studies made on any given case of coma we frequently find that the diagnostic procedure also becomes good therapy. It will be seen in the course of our subsequent analysis that not infrequently the diagnosis and therapy are virtually the same.

Even assuming the necessity for an etiologic diagnosis in a case of coma, it must be faced at the outset that not all cases lend themselves to proper diagnosis. Sometimes the wise physician must stand aside for the march of time to clear the picture. During this period, however, the same diagnostic procedures should be adopted as would apply to any other kind of a case. Despite the fact that the patient is in coma, all available history obtainable from family or friends should be accumulated. At times this may mean considerable effort on the part of the physician, but the results usually are worth the effort. Emphasis should be placed on this point because the natural tendency in a case of coma is to act. As an illustration of the value of obtaining a history, one may cite the case of a woman picked up in a department store and brought into a hospital apparently with hemiplegia. Without any clue obtainable from her it was impossible to make a diagnosis until the family arrived and informed the physician

that she was a patient of one of us (S S ) and taking insulin When we were called in the next day we found that instead of taking 8 units of insulin she had taken 80

The alcohol breath which may be found in so many cases of coma is in itself of little value, but if an attendant makes the statement that the patient never drank or the opposite statement, that the patient is a confirmed drunkard, much diagnostic and therapeutic help is obtained

Again, before attempting any therapy a most careful physical examination is indicated, and even at the risk of emphasizing the obvious, we believe it necessary to stress the extreme value of accurate observation, particularly of the skin, mucous membrane, and the type of breathing The diagnosis may be made in certain cases of come by the presence or absence of cyanosis, Cheyne-Stokes breathing, the Kussmaul breathing or a slow or shallow breathing Signs of external injury demonstrable by blood coming from the nose, mouth, eyes or ears, injury to the scalp, and the signs of trauma anywhere else in the body must be carefully looked for The odor of the breath, of which we have previously spoken, may or may not be of importance but must be carefully noted It is not always possible particularly in the presence of mouth infection to distinguish types of odor in the breath

A study of the reflexes and of muscle tone and all the other tests which go to make a good neurologic examination are of course indicated Whatever is found from this examination must be carefully analyzed The absence of reflexes, for instance, may be the result of shock rather than a sign of prime importance Pin-point pupils of opium poisoning as contrasted to the dilated pupils associated with barbitol poisoning may form a decisive point in differential diagnosis The finding of an enlarged heart and fibrillating pulse in the case of coma suggests naturally a cerebral embolus, and a fast feeble pulse may indicate the failing heart of hemorrhage, coronary disease, or may be a terminal finding

In the treatment of a case of coma the presence of sugar, acetone or albumin is of obvious significance, but it should be

emphasized that no one of these substances is absolutely specific. Sugar may occur not only in diabetes, but in almost any type of brain injury. Acetone while it may indicate diabetic acidosis, especially when associated with other clinical signs, may occur in starvation and be of absolutely no diagnostic value. Albuminuria likewise cannot be interpreted entirely in association with other physical findings. The urine in all cases of coma should be saved so that if there is other evidence to suggest poison, the specific poison may be looked for.

Interpretation of the blood pressure will depend to a great extent on any information obtainable concerning the preceding state of the blood pressure. If a patient is seen with peripheral arteriosclerosis and a low pressure and we are told by the family that he previously had a high blood pressure, we immediately must suspect a cerebral accident.

No better illustration of the point mentioned earlier regarding the relationship between diagnosis and therapy can be cited than gastric lavage. Perhaps done primarily for diagnostic purposes, the diagnostic test immediately becomes a therapeutic procedure.

It is our belief that lumbar puncture may be used as a routine procedure, either diagnostically or therapeutically, and may clear up the whole picture.

From a therapeutic point of view coma cases may be divided into four groups: (1) Those requiring immediate trephining, (2) those requiring stomach lavage and antidotes for drugs or other poisons, (3) those requiring specific treatment as in diabetic coma or a real case of poisoning, and (4) those requiring rest and watchful waiting.

Fortunately only a few cases require immediate trephining and they are the ones which show evidence of increased intracranial pressure, such as increase in blood pressure, with slow pulse, nausea and vomiting, and choked disk. Such cases usually are either traumatic or may be due to brain tumor, and usually a trephining operation is then performed. If the increased pressure is due to cerebral hemorrhage and if this



hemorrhage arises from the middle meningeal artery, this artery may be ligated

The first treatment of cases due to poisoning is lavage of the stomach. If the specific drug can be found antidotes should immediately be given. In cases of morphine and barbitol poisoning, potassium permanganate is used in the lavage solution. If mercury is the etiologic factor milk or egg-white should be used.

Coal gas poisoning is usually identified by the cherry red color of the patient and specific changes in the spectroscopic bands. Treatment by oxygen and carbon dioxide inhalations is urgent, and according to some, intravenous injections of methylene blue may be helpful.

In not all cases can a specific drug be identified and certain general measures are indicated. A strong cathartic, such as Epsom salts or castor oil, should be given after the lavage. Stimulants, such as caffeine, strychnine and coramine, may be of urgent need. Intravenous glucose is a rapid method of stimulating urinary secretion. This may be used in 5 to 10 per cent solution or even stronger. In all cases the patient should be kept warm by blankets, hot-water bags or electric pad, and should be turned frequently from side to side. Artificial respiration may be compulsory in some cases. The feeding problem is not easy but an effort should be made to maintain nourishment. Rectal feeding is not very satisfactory but has to be used. A nasal catheter may have to be used, especially in cases of poisoning from caustic drugs. The nasal catheter is generally speaking the best means at our command at present.

In the third group of cases, namely, those with a specific etiology, we find our most satisfactory results in diabetic coma. In this condition it is necessary constantly to keep in mind the principle for which one is striving. For instance, the height of the blood sugar or even the exact amount found in the urine is not important because the coma depends on acetone bodies rather than on glycosuria. In the treatment of diabetic coma insulin is a specific and provided there are no

complications will in the majority of cases result in restoring the patient to consciousness. Although insulin can never be given blindly, it is always safe to give a comparatively large dose in a proved case of diabetic coma, and in general practice where the controls may not be as accurate as they would be in an institution, we believe it is safer to combine insulin with glucose in some form. To be more specific, the initial dose may be anywhere from 30 to 50 or 60 units without bothering about extra glucose. More than 60 units at a single dose is of little value. The patient's clinical condition is watched and the urine studied, particularly from the standpoint of sugar and acetone. The further treatment is determined by the course of events. The urine should be examined every two hours, even if it is necessary to catheterize the patient. If at the end of the first two hours there are signs of diminishing acidosis the amount of insulin may be reduced to 20 to 40 units, but at this time we believe it is wise to add glucose either in the form of hypodermoclysis (2.5 per cent solution) or by mouth if the patient is now able to drink orange juice or milk. As the acidosis clears up the insulin is further reduced. If despite large initial doses there are no signs of improvement more insulin should be used, but this should be controlled not only by urinalysis but by blood sugar studies. The careless use of insulin must be distinguished from the use of large doses, because the careless use of too large doses may result in hypoglycemia and coma due to this condition. We have seen this happen and we believe that it is a fairly common occurrence which must be guarded against.

General measures are of extreme importance in diabetic coma. The patient must be kept warm, the bowels should be emptied by a cleansing enema, and particularly, dehydration, which is so commonly a concomitant symptom, must be combated. Hypodermoclysis should be instituted immediately and should be continued until the dehydration is controlled. The heart bears a terrific burden and must constantly be watched, particularly in older people. When the treatment of the dehydration does not improve the cardiac condition,

most drastic treatment is indicated. During and immediately after the coma no great attention need be paid to the control of the diet, since carbohydrates are definitely indicated during this period. However, as soon as the patient passes the borderline, the usual principles of dietetic therapy are applicable.

In contrast to the dramatic specific therapy of diabetic coma, the case of uremic poisoning offers only certain general therapeutic principles. Perhaps the intravenous injection of glucose solution, 200 to 300 cc of a 10 to 25 per cent solution at intervals of a few hours, offers the best therapeutic hope. Small but repeated transfusions preceded perhaps by venesection have given good results in some hands. The use of stimulating drugs, such as digitalis or caffeine, are certainly indicated when the heart or peripheral circulation fails. Some investigators have claimed rather brilliant results from the use of intravenous solution of 40 per cent urea given in 50 cc doses every eight hours. We have not seen as good results from this procedure as have been reported in the literature.

In the fourth group belong practically all cases of coma due to accidents to the cerebral blood vessels. Much has been written about the diagnosis and treatment of these conditions. Our own experience suggests that at times it is almost impossible to differentiate cerebral hemorrhage from thrombosis and a careful analysis of our results suggests that the less interfering therapy is used the better the prognosis. At the onset it is impossible to predict the outcome. What looks like a mild cerebral accident may progress steadily downhill, and what starts off as a wild outburst may quiet down. It has been our policy not to do a venesection, lumbar puncture, or move the patient. We believe that the patient should be kept where he is whenever possible, at least for the first twenty-four hours, and nothing whatever but careful watching and nursing is indicated. In the presence of extreme restlessness or convulsive seizures morphine or opium derivative is indicated to obtain the essential quiet and rest. After the first twenty-four hours the general condition of the patient requires

attention Dehydration is to be avoided The bowels must receive attention and, if necessary, stimulation by caffeine or digitalis given

Not all possible causes of coma have been included in this discussion nor has anything been said about the problems involved when the patient has one disease and gets coma from something else The diabetic may go on an alcohol spree and when he is seen by the doctor it is not an easy matter to decide whether he is suffering from alcoholism or from diabetic coma The patient with a known duodenal ulcer may take an overdose of a drug and it will require keen diagnostic judgment to decide whether the coma is due to cerebral anemia from hemorrhage or to a drug The type of case which will be most common in one locality or one institution may be less common in another Alcoholism, for instance, is very rare at Michael Reese Hospital, while in the public institutions it is apt to be one of the commonest causes of coma



## CLINIC OF DR WILLIAM A. BRAMS

### MICHAEL REESE HOSPITAL

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#### VENOUS PRESSURE

EVERY clinician is familiar with the close relationship which exists between arterial blood pressure and myocardial function of the left side of the heart. It is not so generally recognized that venous pressure is a very reliable index of functional capacity of the right side of the heart with the result that estimation of pressure in the peripheral veins has not as yet become a popular procedure in clinical medicine. It cannot be said that determination of venous pressure is a recent development, for Stephen Hales in 1733 reported his experiments in these words "In December I laid a common Field Gate on the Ground, with some Straw upon it, on which a white Mare was cast on her right side, and in that Posture bound fast to the gate, she was fourteen Hands and three inches high, lean, tho' not to a great Degree, and about ten or twelve Years old. Then laying open the left Jugular Vein, I fixed to that part of it which comes from the Head, a Glass Tube, which was four Feet, and two Inches long. The Blood rose in it, in three or four Seconds of Time, about a Foot, and then was stationary for two or three Seconds, etc., etc."

Nor can it be said that modern clinical methods for estimating venous pressure are difficult or inexact. Practically all procedures may be classified as either indirect or direct. Most of the indirect methods are based on the principle of compressing a visible, superficial vein in the skin by means of a transparent air chamber and reading off on a water manometer the pressure necessary to obliterate completely the vein under examination. These methods are used widely and are easy to perform but considerable experience is needed to recog-

nize the end point of compression, a change which is not always sharply defined

We prefer a direct method because it is more accurate and the end point is always distinct. A simple instrument designed by Dr J S Golden and myself overcomes the usual objections raised against direct methods, namely, that the hollow needle may become plugged by a clot and that prolonged observations are impractical. Our apparatus consists of a small metal reservoir and a water manometer, both being connected to a small hollow needle by a glass T tube and suitable rubber tubing. The entire instrument is carried in a small metal box, in which it may be autoclaved and both the reservoir and manometer may be easily attached to the opened cover when readings are to be made. Sterile 3 per cent sodium citrate solution is poured into the reservoir and manometer and the fluid is allowed to fill the tubing and needle, care being taken to expel all air bubbles. The patient is kept in a recumbent or semirecumbent position with the arm well abducted for at least fifteen minutes and the level of the auricle is marked on the side of the thorax according to the suggestion of Eyster. The hollow needle is then introduced into the cubital vein and citrate solution from the manometer tube is permitted to flow into the vein until the level of the solution in the manometer tube becomes stabilized. The height of this level above that of the auricle represents the peripheral venous pressure. The apparatus may be maintained in faultless working order for three to four hours without discomfort to the patient and without removal of the needle from the vein by permitting a few drops of citrate solution from the reservoir to flow through the needle into the vein every five minutes.

We have adopted a range of 5 to 10 cc of citrate solution above the level of the auricle as the normal venous pressure when taken according to our method. Abnormal variations are found in many conditions, but we shall briefly describe our clinical experience with venous pressure in cardiac failure and with certain therapeutic measures used in combating this condition.

It must be stated at the outset that an abnormal level of venous pressure is not a sign of any particular valvular defect or special anatomical lesion. It does furnish very important information as to the myocardial capacity of the right side of the heart. Any condition which leads to failure of the right side of the heart causes a rise in peripheral venous pressure but so-called "pure, left sided failure" causes no such change. Hence, the procedure is of value in distinguishing these two forms of failure in which the treatment is also different. It is of further advantage to estimate venous pressure in instances of right sided failure because cyanosis and the apparent size of the cervical veins may at times be misleading. For instance, much more carbon dioxide in the blood and oxygen deficiency will be necessary in cases of anemia to produce a given degree of cyanosis than in normal persons and much less in patients with an excessive number of red blood cells. This is merely an example of the extracardiac factors which may have an important relation to the degree of cyanosis. We have observed that the apparent degree of engorgement of the cervical veins may be deceptive. Large cervical veins may be due chiefly to emaciation or to local obstruction. Measurement of venous pressure in such instances will furnish much more reliable information than mere inspection.

Elevation of venous pressure in a patient known to have cardiac disease but who is apparently in a good state of compensation will sometimes serve as a warning of impending failure. It may, in fact, be the earliest sign of impending cardiac defeat in a patient in whom such a state is not suspected. The information gained by repeatedly estimating venous pressure may be of value in following the progress of right sided failure. Tendency of venous pressure to fall usually means improvement while persistence of a high level or tendency to rise frequently presages complete cardiac defeat. The results of treatment may also be followed by such repeated observations and great value is placed by some on estimation of venous pressure as a guide for the performance of venesection in instances where venous pressure is high. Dr



J S Golden and I were interested in the value of so-called "bloodless venesection" in patients with heart failure and high venous pressure. As is well known, the procedure consists of constricting all four limbs near the trunk, sufficient compression being applied to permit inflow of blood into the limb while outflow via the veins is prevented. It was thus hoped to relieve the heart of a certain quantity of blood by imprisoning blood in the limbs and to accomplish this without actual loss of blood, since hemorrhage is not always well borne by patients with heart disease. We permitted the constrictors to remain in position for thirty minutes after control readings were taken and measured venous pressure in one unobstructed limb at intervals of five minutes. Blood pressure was also measured and the pulse rate counted at the same intervals. We could find no change in venous or arterial pressure or pulse rate, although the same patients showed an immediate and partially sustained drop in venous pressure after ordinary blood letting. We have, consequently, omitted this procedure from our therapeutic armamentarium.

Dr Golden and I performed a series of experiments to determine the effect of ordinary blood letting on patients with varying degrees of cardiac failure with and without elevated venous pressure. We found no change in the pulse rate and only a transient fall in arterial blood pressure, but venous pressure fell in every instance. The fall in venous pressure was marked in many instances but nearly always returned about halfway toward the previous level within an hour. We have watched the ultimate course of these patients and believe that venesection may be of benefit in some cases of right sided failure with high venous pressure, but we cannot share in the enthusiasm of ardent advocates of venesection. We have followed many patients after venesection and are convinced that more conservative measures would have been of nearly equal benefit. It is possible that our patients had very little cardiac reserve, but we are inclined to rely on digitalis, sedatives and diuretics and to advise venesection only occasionally in right sided cardiac failure.

## CLINIC OF DR HEYWORTH N SANFORD

### PRESBYTERIAN HOSPITAL

#### THE IMMUNIZATION OF INFANTS AND YOUNG CHILDREN AGAINST INFECTIOUS DISEASES

ONE of the most important fields of pediatric procedure exists in the immunization of infants and children against infectious diseases. This has been so popularized among the general public that at the present time most parents are more than anxious to cooperate with the physician on these measures. It is well, however, to realize that not all infectious diseases can be prevented by definite control measures. Unless this is made clear to anxious parents, extravagant promises of disease prevention will cause doubts to arise in the public's mind that will destroy all confidence in preventive pediatrics. It is well, therefore, to divide these diseases into two groups. First, those in which we have a definite method of prevention and immunization, and second, those in which we have a partial or limited method of control.

#### THOSE COMMUNICABLE DISEASES FOR WHICH THERE IS A DEFINITE METHOD OF PREVENTION AND IMMUNIZATION

**Smallpox.**—During epidemics of smallpox, vaccination should be performed at any age. This includes infants in the newborn period. As a general public health procedure, infants should be vaccinated from the sixth to the twelfth month of life, or not later than the eighteenth month of life. The reasons for doing so at this time are that they are given protection from the disease (there is hardly any inherited immunity), the reactions are almost negligible at this age, and lastly, post-vaccinal encephalitis hardly ever occurs under eighteen months of age.

In the absence of an epidemic, it is usually well to postpone vaccinating children afflicted with such skin diseases as eczema, impetigo, and furunculosis. Infants suffering from severe gastro-intestinal upsets, or marked malnutrition may be spared the added burden of vaccination until they are stronger. While it is probably quite hypothetical, I believe that the best time for vaccination is in the late spring, early summer and fall. Obviously the hot months are depressing and excessive perspiration may cause scratching of the pustule and secondary infection. During the winter and early spring the child may suffer from a respiratory infection or contagious disease. While most parents are intelligent enough to know that these diseases are not caused by the vaccination, still in the back of their minds they will wonder if the vaccination did not lower the child's resistance to the disease.

Vaccination may be done on any skin surface. The preferred site in boys is at the point over the insertion of the deltoid muscle on the left arm. In girls, the outer aspects of the left thigh, at a point two thirds of the way from the knee to the hip is the best location. One is accustomed to looking for a scar in these locations in examinations, and there is no reason for placing the vaccination in bizarre localities.

The site of vaccination should be thoroughly washed with acetone. Other antiseptics may destroy the virus. A drop of virus is placed in the center of the cleansed area. Holding a small sterile needle at an angle of 20 degrees from the skin surface, the point of the needle is pressed into the skin through the drop about ten times. Obviously these pressure points should be in the same locality. No blood or serum should be drawn. The excess of virus is now wiped off with a piece of sterile gauze.

There is some difference of opinion as to the advisability of putting a dressing over the inoculation. It is permissible to use no dressing. Certainly tight constricting bandages and shields should be strictly avoided. I always put one thickness of sterile gauze over the point of inoculation very loosely with a piece of tape. The mother is told to remove this next morn-

ing and put nothing more on it until the pustule appears. She is then told to place a similar piece of gauze on the pustule, and to change it daily unless the pustule breaks and sticks to the gauze. If this happens the gauze is not removed. No tub bath is given the child from the time the pustule appears until the scab is on firmly. The mother is warned that a papule usually appears on the third to fifth day. In another day this becomes a vesicle and the next day a pustule with a swollen red area around it. At about the sixth day there is frequently some fever and loss of appetite. Mothers are told to report one week after vaccination for inspection and further instructions.

Reactions will fall into three groups. Successful primary vaccinations in which a pustule is present on the seventh day, an unsuccessful vaccination shows no reaction to the virus and confers no protection against smallpox. Those who fail to react after a primary vaccination with the production of a pustule should be revaccinated after two weeks. Children revaccinated after a previously successful result may show a reddened, inflamed macular or maculopapular area at the vaccination site at about forty eight hours. This is evidence of persistent immunity from the previous vaccination.

The complications of vaccination are secondary infection, generalized vaccina, autovaccination and postvaccinal encephalitis. If the procedure is carried out as outlined, secondary infections are very uncommon. They were more common when shields and tight bandages were used. They can be told by excessive swelling, abscess formations and lymphatic enlargement. Secondary infection of the pustule may be treated by cleansing the pustule with ether and painting with a mild antiseptic as gentian violet or mercurochrome. Abscesses should be treated surgically with wet dressings.

Generalized vaccina occasionally happens. It varies from scattered pustules around the primary lesion to scattered papules and vesicles over the entire body. The child may have several degrees of temperature and be quite prostrated. There is nothing to do for these children except to give anti

pyretics for the temperature The prognosis is excellent and recovery rapid Those children who have eczema or delicate skins appear more likely to have such reactions

Autovaccination is caused by not wiping off the excess of vaccine which the child scratches and carries to other parts of the body, or by scratching an uncovered pustule The most common points are the lips, cheeks or eyelids They follow the course of the original vaccination Nothing can be done and they are unimportant with the exception of the eyelid or cornea This is very serious and may cause loss of the eye Call an ophthalmologist into consultation immediately if this happens

Postvaccinal encephalitis has received considerable attention abroad In this country only 75 cases have been reported Considering the number of antismallpox vaccinations performed yearly, this number is very small In the countries where postvaccinal encephalitis occurred, it affected children mostly of school age, only rarely has a case occurred in infants under two years An exception is Germany, where nearly all cases are between one and two years However, most children in Germany are vaccinated at that time

The symptoms appear suddenly, on the tenth to the thirteenth day following vaccination Fever of  $104^{\circ}$  F, vomiting, headache, stupor and positive Babinsky are the most important symptoms The course is stormy and the prognosis is grave The mortality is 40 per cent Recovery, when it takes place, is usually rapid and complete Besides the usual treatment for encephalitis, the child should be given blood serum from a recently vaccinated person

As a preventive measure for vaccine encephalitis, the following principles should be used in vaccination Primary vaccination should be done early in life, during the first year if possible No person should be vaccinated unless in perfect health Mass vaccinations are to be avoided In Holland, most cases of postvaccinal encephalitis occurred in March and April Multiple scarification and cross hatching were condemned by the English committee Armstrong recom-

mends that primary vaccinations, especially after the first year of life, be deferred until immunization against diphtheria or other diseases has been accomplished, as this preliminary exercise of the immunity or defense forces may lead to a more efficient antiviral response

**Diphtheria**—Immunization against diphtheria should be a routine procedure. Most infants have a natural immunity to diphtheria at birth that begins to fall off at about the sixth month. For this reason it has been advocated that immunization should be begun at this time. Certainly in infants living in slum surroundings, more liable to infectious contacts, this is advisable. In the average infant that has medical supervision, I do not think it is advisable to immunize before one year of age, but it should be routine at this time. Certainly in babies of this type, diphtheria under eighteen months of age is quite uncommon. Furthermore, the work of Greengard indicates that the passive immunity in young infants interferes with the development of antitoxin in response to vaccination with diphtheria toxoid. The only infant in my experience that died of diphtheria under fifteen months of age had received toxoid at six months of life.

Immunization should be routine at one year and advocated up to ten years. A Schick test preliminary to immunization is unnecessary for children less than seven years of age. For older children it is desirable to determine the need for immunization.

Toxoid should be employed for children under seven years of age. At the present time most men are using two doses of 1 cc. each, at not less than two nor more than four week intervals. Some give three doses which probably gives a higher immunity, but inasmuch as two doses of 1 cc. each at two- to four-week intervals give a 90 to 95 per cent immunity, this hardly seems necessary. The one dose alum precipitated toxoid in amounts of 0.5 to 1 cc. (varying with different preparations) may supplant the other toxoid. It has the advantage of one injection because of slow absorption. It appears to be very successful in children under seven years

of age It should be used with great care in older children because of local reactions

The arm over the deltoid region is cleansed with soap, water and alcohol The toxoid is given in a sterile syringe and needle subcutaneously Be sure that there are no air bubbles in the syringe, and that the needle is wiped dry, also do not use formaldehyde toxoid solution unless it is perfectly clear The injection site is covered with a sterile gauze bandage, removed the next morning The mother can be assured that there will be no reaction at this age The second injection is given in the other arm in two to four weeks

Immunization with toxoid should never be done for a child of over seven years without first testing their reaction to it This is done by injecting 0.1 cc of toxoid intradermally If within three days an area of redness measuring more than 10 mm develops, either one of two procedures may be used The first is to use toxoid, giving one dose of 0.2 cc subcutaneously followed in two weeks by a second dose of 0.5 cc and in two more weeks by a third dose of 1 cc In the event of a reaction after the first dose, the amounts subsequently injected should be cautiously increased or the same dosage repeated Immunity may be produced by three injections of as little as 0.2 cc each The second method is to use toxin-antitoxin mixture, which generally produces less reaction in older individuals Three successive injections of toxin-antitoxin mixture, in amounts of 1 cc each, can be given subcutaneously, from one to two weeks apart

Diphtheria immunization is worthless unless it is followed up with the Schick test While with the two-dose method of toxoid, 90 to 95 per cent of children will become immune within three months, the five or ten children who are not immune could spoil the effect of the entire procedure by raising doubts in the mother's mind as to the reliability of the method

Diphtheria toxin for the Schick test is supplied in ten test ampules The ampules are 1 cc each and contain diluted toxin in peptone solution They are stable for a period of months, as stamped on the bottle, if precautions are used

These consist in keeping the bottle in the ice-box when not in use, and thoroughly wiping off the rubber top with alcohol before piercing with the needle. Obviously the needle and syringe should be sterile. Use a syringe graduated in 0.1 cc. and a fine needle (26 gauge) with a rounded point. Cleanse the flexor surface of the arm with soap and water and alcohol and inject 0.1 cc. of the diluted toxoid intradermally. A superficial bleb should result, through which the hair follicles can be seen. Cover this with a piece of sterile gauze, to be removed the next morning.

The test is read in forty-eight to seventy-two hours. A positive reaction is shown by redness of the skin covering an area of over 10 mm. in diameter. Positive reactions usually stay red for several days, turn brown and scale over the point of injection. A negative reaction shows a red area less than 10 mm. Schick tests have been made on so many children with such conformity of results, that the mother can be assured that her child is now immune to diphtheria. As a further safeguard, it should be recommended that the child be re-Schicked on entering school, or in the presence of an epidemic. Some children have been known to lose their immunity in four or five years. If a positive reaction is obtained, one may wait another three months and repeat the Schick test, or give another 1 cc. of toxoid at once, and re-Schick at three months. Some children will no doubt become negative in another three months, but I always give one more dose of toxoid to be safe. Schick reactions are uniformly negative after the third dose of toxoid.

It must be emphasized that immunization does not take place for three months and none of these methods should be relied upon for active immunity in the presence of diphtheria. In such cases 1000 units of diphtheria antitoxin should be given intramuscularly. Remember that this is a horse serum and the child may be sensitive to it. All children with a history of eczema, asthma, or any sensitivity, or who have had any horse serum before (including toxin antitoxin mixtures) should be tested for sensitivity before giving the injection, by injecting intracutaneously 0.1 cc. of a  $\frac{1}{10}$  or  $\frac{1}{100}$  dilution



of antitoxin If no wheal or marked zone of erythema appears within thirty minutes, the full dose may be given If such a reaction appears, the antitoxin should be given in divided amounts beginning with 1 cc in  $\frac{1}{100}$  dilution with saline solution At intervals of thirty minutes increasing amounts may be given until the entire amount is completed This should be given in an extremity, so that rapid absorption can be impeded by a tourniquet if an immediate reaction issues In case of a severe reaction, 3 to 6 minims of a  $\frac{1}{1000}$  epinephrine solution should be given subcutaneously

**Typhoid Fever**—While immunization against typhoid fever is a definite prophylactic procedure, it is not necessary in most communities for universal application Its use should be restricted to those communities where typhoid incidence is great and particularly if a trip is planned to such a region This applies particularly to Europe and the Orient, or to rural communities in this country where the water supply is questionable

Immunization could probably be given to the child at any age Certainly at one year of life it would be satisfactory The vaccine given for adults is in amounts of 0.5 cc for the first dose and 1 cc for the second and third doses There is no definite amount recommended for children I usually give children over eight a full adult dose and under eight years two thirds of the adult dose The injections are given subcutaneously in the deltoid region at one-week intervals As a rule, children have no reactions It probably takes about three months to establish an immunity There is no simple laboratory test for determining typhoid immunity, but it is assumed that immunity lasts at least two years If protection is to be assured beyond this time, a second course of injections should be given, or one dose may be given each year following the original course of three

**Tetanus**—Tetanus antitoxin should be always used prophylactically in all cases in which a wound is contaminated with horse manure or human excreta This, therefore, includes all wounds of the street, in gardens, or any spot where

street dirt may have been trampled. Also all wounds contaminated with water at bathing beaches, or industrial injuries. It is particularly indicated in puncture wounds or wounds about the head. The prophylactic dose is 1500 units injected subcutaneously. Remember that this is a horse serum and the precaution mentioned under diphtheria should be followed.

At the present time there is much work being done on vaccination against tetanus, which may develop into a standard procedure later. Ramon has been using an anatoxin mixture in French soldiers with apparent success. Bergey and Etries in this country recommend one injection of a 1 cc. alum precipitated tetanus toxoid for immunization. This is given in one injection and gives an immunity in three to six months. They claim there are no reactions. At present, however, only antitoxin should be relied on.

#### THOSE DISEASES IN WHICH PREVENTATIVE MEASURES ARE LIMITED OR INDEFINITE

In these diseases the mother should be told that the methods of immunization are not perfect. It is folly and detrimental to the public faith in preventive pediatrics to make extravagant claims as to immunizing children for these conditions.

**Scarlet Fever**—Natural immunity is found in many children, and a considerable number of adults. Inherited immunity is present in the newborn if the mother is immune, and lasts in one half the children for six months and in one third for twelve months. Clinically scarlet fever is uncommon under two years. For this reason infants may be Dick tested at any time, preferably after one year. I test routinely between eighteen months and two years.

Dick test material is obtainable in 10-test ampules and 100 test ampules. The 10-test ampules are glass topped and must be used at once. The 100 test ampules have a rubber top and if care is used as outlined with Schick test material (use acetone to sterilize top, not alcohol), they may be used until the expiration period stamped on the bottle. Great care

must be used in performing the Dick test. Use a syringe graduated in 0.1 cc. and a 26-gauge needle with a round end. Boil the syringe and needle in distilled water. Use no alcohol. Cleanse the skin with acetone. Expel at least 0.1 cc. of the scarlet fever toxin from the needle before using. Inject accurately 0.1 cc. of the scarlet fever toxin intradermally. Injections must be intradermal as a subcutaneous injection may show negative results in a positive subject. Cover with a sterile gauze bandage and read in twenty to twenty-four hours (not later). Unlike the Schick test there will always be some erythema. If the area of redness measures over 10 mm. in diameter, the test is positive.

If the test is negative, it may be assumed that the child is immune to scarlet fever. However, it must be remembered that this test is not perfectly reliable. Persons with negative Dick tests have been known to contract scarlet fever. The mother should be warned of this and told to have the child retested in the presence of a scarlet fever contact, or epidemic or in any case in two or three years.

If the test is positive, all of the factors that enter into the situation should be explained to the mother. Nursing groups, personnel of contagious hospitals and orphanages, or groups intimately exposed to scarlet fever should be protected. The Committee on Prophylactic Procedures Against Communicable Diseases of the American Academy of Pediatrics does not recommend active immunization by scarlet fever streptococcic toxin as a general public health procedure because local and general reactions are frequent in adults and not altogether absent in children. Also the degree and duration of the immunity have not been definitely established. However, the degree of reaction is certainly not as bad as a case of scarlet fever. I believe that the principal argument against it is the degree and duration of immunity. These facts should all be laid before the mother and let her decide. I certainly would not force the immunization against her desire not to have it.

Active immunization, if decided upon, consists in the injection of five increasing doses of scarlet fever streptococcus

toxin at weekly intervals. These injections are made subcutaneously as with the diphtheria toxoid. This material is put up in 1-cc vials and are numbered one to five. Great care must be taken to give these doses in order as they increase rapidly in toxin and would cause serious reactions if given out of order.

It is stated that about 10 per cent of susceptibles will show general reactions after the first dose and about the same number following the last dose. From my own experience, I would say that 30 per cent was a closer figure. These reactions are a temperature up to  $102^{\circ}$  F, vomiting, diarrhea, and headache. These come on in a few hours and last about twenty-four hours. They may be treated symptomatically. I find the vomiting most troublesome. It may be necessary to withhold all food and give only tablespoonfuls of water for twenty-four hours to control it. In the presence of severe reactions it is best to divide the next dose of toxin, and give it the following week. If there are no reactions, the usual routine can then be followed.

Two weeks after the last dose of toxin, repeat the Dick test with 0.1 cc of toxin in the right arm and 0.2 cc. in the left arm. If either test is positive, repeat the fifth dose, after which 95 per cent of the susceptibles will become negative. The mother should be instructed to have the Dick test repeated again in one or two years, or in the presence of a contact or epidemic.

This active immunization, of course, takes at least five weeks and is valueless for a susceptible contact. For these children, convalescent scarlet fever serum has been recommended for passive protection. It is given intramuscularly in amounts of 10 to 20 cc. The evidence as to its reliability is inadequate, but it should cause no reactions and I think should be used if obtainable. Scarlet fever antitoxin is also used for passive immunization. Its use is frequently accompanied by unpleasant horse serum reactions and the evidence regarding its value is indefinite.

**Measles**—There is no method for active protection

against measles The disease may be prevented, or better, modified by the injection of blood serum of a person recently convalescent from the disease, or the blood serum of adults

Human blood serum taken one to three weeks after the fever of measles has subsided should be used When administered within five days after exposure in doses of 10 cc intramuscularly it may protect against the disease It usually lessens the severity of the disease in any case and should be used in children under three years of age, or those suffering from another disease, particularly a respiratory infection Under ordinary circumstances, it is more desirable to produce modified measles than to protect completely Ten cc of convalescent serum given six to seven days after the child has been exposed produces partial protection That is, measles develops in a mild form

Thirty cc of normal adult whole blood, or 20 cc of normal adult serum, will produce somewhat the same effect as 10 cc of convalescent serum It probably is not as reliable due to differences in protective substances Measles modified by convalescent serum or normal blood apparently produces a permanent immunity comparable to the disease McKahn has advocated the use of placental extract It is believed that 2 cc injected on the sixth or seventh day after exposure will usually modify the disease This method could be used in the absence of convalescent serum, or adult serum

**Whooping Cough**—A vaccine for the active immunization against whooping cough has been developed by Sauer This vaccine differs from the older types in that it is prepared from fresh strongly hemolytic strains of *B pertussis*, grown on medium made with freshly defibrinated human blood This gives the advantage of more massive doses than with the older vaccines It apparently gives considerable immunity Sauer believes that the best age to give it is between the seventh and tenth months If the child has been immunized against diphtheria or smallpox, several months should elapse before pertussis immunization

It should be remembered that more evidence should be gathered on this procedure before it is adopted as a general

public health measure Whooping cough is one disease for which the infant has no natural immunity almost all of the mortality occurs in the first and second years of life, therefore, if the infant can be protected, it is highly desirable It takes four months for immunity to develop from Sauer's vaccine, therefore, it would be necessary to give it quite early in life Whether it is wise to immunize young infants in the face of possible reactions remains to be seen

In regard to older children, or those younger who have been immunized against diphtheria and smallpox, it could be considered The facts should be laid before the mother, that reactions occur, and that while probably the majority of children will become immune, there is no test at the present time to discover who has become immune and who has not Therefore, immunity cannot be guaranteed As it takes four months for immunity to develop, immunizations should not be given during an epidemic

The injections are given subcutaneously at weekly intervals for three weeks The initial dose is 1 cc. in each deltoid, the second 1.5 cc in each triceps, and the third 1.5 cc in each biceps This makes a total dosage of 3 cc The skin should be cleansed with soap, water and alcohol, and the syringe and needle boiled in distilled water for ten minutes

Reactions when they occur may be general or local with a rise in temperature to  $101^{\circ}$  F four to forty eight hours after injection, and tenderness, redness and induration Movable, subcutaneous nodules of induration may exist for a month Usually no treatment of any kind is necessary nor any change in routine of injection

There are several methods of prophylactic immunization employed for those exposed to the disease The older vaccines containing 5 to 10 billion killed organisms per cubic centimeter are given subcutaneously in doses of 0.25, 0.5, 1, and 1.5 cc at intervals of two to three days Their efficiency is under much dispute

Frawley has reported good results in the use of undenatured pertussis antigen for prophylaxis The initial dose is 1 cc given subcutaneously, followed by 2 cc every other

day until 6 doses have been given. Human blood serum is used in the culture medium, and there seems to be very little local or general reaction.

Convalescent serum from patients recovering from whooping cough has been used in amounts of 10 cc. for children known to have been exposed to the disease. The evidence in regard to this is indefinite.

**Chickenpox and Mumps**—Blood serum from recently convalescent individuals has been used for both these diseases. Their value is not absolutely proven and the indication for such use is infrequent due to the mildness of these infections.

**Poliomyelitis**—Kolmer has developed a method of vaccination against poliomyelitis by giving a suspension of monkey spinal cord, from animals receiving an inoculation with sodium ricinolated virus. This is given in three increasing doses at weekly intervals. It has only been used on a few individuals and is not commercially available as yet.

For passive immunity convalescent serum and normal serum have been proposed during epidemics. Convalescent serum might be of value, but it is so limited in amount that it should be confined to the treatment of early cases. Normal serum and whole adult blood have been used in amounts similar to those used in measles immunization. The value of these has not been demonstrated, but they certainly can do no harm.

**Rabies**.—If a child is bitten or scratched by a dog (cats and monkeys can also have rabies), and the skin is broken, give vaccine if the animal is unknown, disappears, dies in less than ten days, is killed in less than ten days, or develops rabies. If it has a suspicious sickness, begin vaccine and observe. It is not necessary to give vaccine if the dog remains well. Contaminated bites should be cauterized with nitric acid. Head wounds are the most dangerous.

Different preparations are supplied, but all are usually emulsified rabbit cord, infected with a fixed killed virus. They are incapable of producing rabies as a result of the injection. The treatment consists of 14 daily injections given subcutaneously. In cases of extensive injury, or more than a suspicion of rabies, 21 to 28 injections should be employed.

## CLINIC OF DRS JESSE R GERSTLEY AND MEYER TEITELBAUM

MICHAEL REESE HOSPITAL AND NORTHWESTERN UNIVERSITY  
MEDICAL SCHOOL

### RICKETS IS THERE A DIETETIC FACTOR?

THIS baby is seven months old. To all appearances he is normal. I will not take time to go over all points in the physical examination but simply note no abnormalities other than the barest suggestion of beading of the ribs. This is no more marked than in many babies pronounced physically normal. He has no craniotables nor Harrison's groove. He holds his head up well and makes very definite efforts at sitting up although he does not accomplish the latter with complete facility. His gums are swollen and one of the lower central incisors is just coming through. Possibly in development he may be a few weeks behind what is considered a perfect average. However the fact that he has been boarded in the hospital ever since his birth may be the explanation. Are these findings sufficient to justify a diagnosis of mild rickets?

The family and past histories are negative.

In previous clinics I have discussed "The Normal Infant" and also "Infant Nutrition." Do not be alarmed. This is no repetition. This baby is being demonstrated as an introduction to the subject of rickets.

During recent years the profession and the public have been almost overwhelmed with literature on rickets. I must confess that I do not quite understand the reason. During my student days when there were no known vitamins, rickets did not assume such dread significance, and it is only recently that we find the term on the lips of every physician and layman. Is it possible that in the background lies the propaganda of interested industries? Or is there really an increase in the incidence of the disease?



I am not going to review the subject this morning in the classical style of conventional etiology, pathology and symptomatology. You are familiar with the present-day tendencies. One must penetrate deep into the wilderness indeed to escape the blaring radio and the bombardment of leaflets extolling the virtues of vitamin D as it exists in cod liver oil, halibut liver oil, salmon liver oil, irradiated ergosterol (i.e., viosterol), milk of cows receiving irradiated yeast in their fodder, and irradiated milk. I will not even discuss the recent findings of Bills of the Mead Johnson Company describing differences in the vitamin D action of cod liver oil and the oil of the tuna. There is still much scientific work to be done. Today I want to discuss the subject from a purely clinical standpoint with special reference to some of my thoughts and observations during the last five years.

Probably every pediatrician has noted that some babies develop signs of rickets in spite of receiving supposedly adequate amounts of vitamin D. These findings are usually ignored and the parents assured that the baby is perfect. I have seen little reference to this phase of the subject in scientific literature. One hears more of it in the "off the record" conversations at the medical meetings.

In view of the tradition that rickets becomes most apparent during periods of rapid growth, I have often asked myself whether the present-day craze for huge babies may not be a hitherto unsuspected factor. In this category does a factor lie in the concentrated high caloric diets now so generally used which may induce a period of unnatural growth? Is it also possible that cod liver oil which on the one hand contains the antirachitic vitamin but on the other increases the size of the subject has a disadvantageous as well as an advantageous effect?

There is also another possibility which for some reason or other has been overlooked by pediatricians, namely the effect of the reaction of the intestine. This latter is the specific study I have been making during the last years and is the one I am going to report today.

In previous publications concerned with infant nutrition we have reported that the addition of 12 per cent lactose to boiled whole cow's milk is generally followed both by an increase in the hydrogen ion concentration and the gram positive flora of the stool. In other words, there is in some respects a trend toward the stool of the breast fed. Other sugars studied failed to give similar results. But on the other hand, babies receiving such concentrated high caloric mixtures developed nutritional disturbance presumably from overfeeding.

In 1926 Bergeim reported experiments in albino rats showing a better absorption of calcium when given with lactose than with other carbohydrates. This he attributed to the increase in intestinal acidity following the use of lactose.

During recent year lactic acid milk and other concentrated foods have shown increasing popularity. The addition of acid to cow's milk unquestionably facilitates gastric digestion, but we have shown that the stool remains alkaline. During these years lactose has been practically discarded in infant feeding. Is it possible that the feedings so generally employed fail to acidify the intestine sufficiently to promote proper calcium absorption and in themselves create an increased demand for antirachitics? Can there be an increased incidence of rickets due not only to the caloric content but also to the chemical nature of the formulae used?

In an effort to solve one phase of this problem we have contrasted the effect of a maltose-dextrin preparation with that of lactose upon the development of rickets when the respective carbohydrates were added in equal quantities to diluted cow's milk. The study has been both clinical and metabolic. The present report concerns the clinical observations on 20 infants.

**Method**—The infants were admitted to the hospital as soon as possible after birth and kept in a separate room with special nursing care. No vitamin D was given in any known form. Feedings consisted of ordinary certified milk with no vitamin D addition, diluted with distilled water and boiled one minute. The carbohydrates used were a maltose-dextrin

preparation, lactose and beta-lactose. As our previous work had been done with mixtures of whole cow's milk with 12 per cent carbohydrate addition, it was first planned to use such a mixture, but in quantities restricted to the caloric needs of the infants. As will be seen, the results in the first two infants precluded further use of this combination. So that mixture was diluted to one half or two thirds strength as indicated by the patient's age. In such dilution the relation of carbohydrate to protein remained unchanged.

Calcium and phosphorus intake and excretion were determined in the usual type of metabolic experiment. These results will be reported in a subsequent publication.

Clinical observations consisted of monthly physical and roentgenological examinations as well as determinations of blood calcium and phosphorus.

These clinical observations were presented at a recent meeting of the Chicago Pediatric Society\* and I will now give them informally.

**Results**—The chart (Fig 158) shows the babies studied with reference to the calendar and the diet. Unfortunately for scientific work we had to take the babies when we could get them. Babies 1 and 2 are the only ones receiving the original mixture of whole milk with 12 per cent carbohydrate addition, one lactose, the other maltose-dextrin. All the others received dilutions of the above mixture with the exception of babies 17 and 18, who received whole cow's milk with no addition during most of the period, and babies 10 and 13 who received breast milk.

In this chart we have graded each baby, considering 10 as perfect. The grades were reached by considering physical, roentgenological and blood chemical examinations as well as the reports and impressions of impartial nurses and house physicians in charge of the infants. It will be seen that in this series the babies on lactose showed a very slight superiority. However, it must be remembered that only 20 babies were studied. Observations were extended over four years.

\* December 18, 1934

and conditions and seasons were not comparable. Also the rating was to a considerable extent subjective. Conclusions should not be definite until the chemical analyses are completed.

While there may have been a slight clinical difference between the babies receiving lactose and those on maltose-dextrin, the striking finding was the low incidence of any appreciable

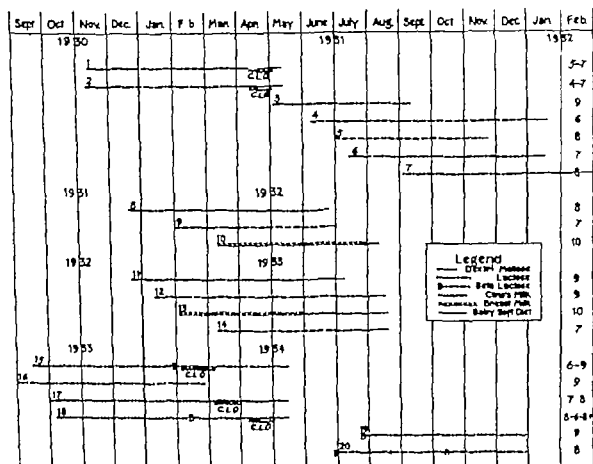


Fig. 138—The cases grouped according to the length of time under observation, the calendar and the diet prescribed.

degree of rickets in either group. Far more important than differences in the type of carbohydrate used was the difference shown between infants receiving cow's milk with high carbohydrate, cow's milk with no addition and cow's milk diluted and with carbohydrate addition. While under no circumstances questioning the importance of vitamin D, one feels that in these findings there is a suggestion of a dietetic element not yet clearly defined.

The chart (Fig 159) shows the findings in baby 13, who received breast milk and served as a control, in babies 17 and 18 who received whole cow's milk and in babies 1 and 2 who received whole cow's milk with 12 per cent carbohydrate. The chart shows the clinical findings as well as the results of monthly x-ray and blood chemical examinations.

The contrast in the findings is startling. It is only fair to state that babies 1 and 2 were twins, came to us somewhat

Baby's No	1st Month	2nd	3rd	4th	5th	6th	7th	8th
13	Ca 12.2 Cr? P 56 XR 0	Ca 11.1 P 51 XR 0	Ca 11.3 P 63 XR 0	Ca 9.6 P 59 XR 0	Ca 11.7 P 64 XR 0	Ca 9.8 P 59 XR 0	Ca 11 Ry? P 58 HG XR 0	
17	Ca 11 P 52 XR 0	Ca 10.3 Ry? P 57 HG? XR?	Ca 10.4 Cr? P 47 Ry? P 50 HG? XR? HG?	Ca 10.5 Ry? P 50 HG? XR?	Ca 10.6 Ry? P 61 HG? XR?	Ca 10.3 Cr? P 53 Ry? P 53 HG? XR? HG?	Ca 10.8 Cr? P 50 Ry? XR? HG?	
18		Ca 10.7 P 64 XR 0	Ca 11.3 Ry? P 59 HG? XR?	Ca 10.9 Cr? P 55 Ry? XR? HG?	Ca 9.2 Ry? P 60 HG? XR?	Ca 9.1 Ry? P 63 HG? XR?	Ca 9.7 Cr? P 50 Ry? XR? HG?	Ca 11.5 Cr? P 53 Ry? XR? HG?
1	Cr? P 32 Ry?	Ca 10 Cr? P 32 Ry? XR+	Ca 6.2 Cr? P 55 Ry? XR+	Ca 8.8 Cr? P 46 Ry? HG+	Ca 8.1 Ry? P 45 Ca 8.4 P 36 XR+	Ca 7.6 Ry? P 32 HG+ XR+	Ca 10.1 Ry? P 61 HG+ XR+	
2	Ry? P 29 Ry?	Ca 9.4 Cr? P 29 Ry? HG?	Ca 7.1 Cr? P 49 Ry? XR+ HG+	Ca 7.2 Cr? P 45 Ry? XR+ HG+	Ca 7.6 Ry? P 31 HG+ XR+ Ca 8.6 P 36	Ca 8.9 Cr? P 36 Ry? XR+ HG+	Ca 9.6 Ry? P 63 HG? XR+	

Legend  
 Cr = Craniotabes  
 Ry = Rosary  
 HG = Harrison's Groove  
 XR = X-Ray  
 ? = Mild  
 ? = Early Definite  
 M = Whole Cow's Milk  
 BL = Beta Lactose

Fig 159—The blood calcium and phosphorus as well as the x-ray findings and the presence of craniotabes and rosary on diets of breast milk, whole cow's milk, and whole cow's milk with 12 per cent carbohydrate

later than the other infants, and were slightly rachitic upon admission. All these factors probably played a part in determining the severity of the rickets which was by far more pronounced than in any of the babies studied. But at any rate there was no spontaneous improvement in the condition. We did not feel justified in repeating the experiment though at the conclusion of the period of observation a brief treatment

with cod liver oil brought the findings rapidly to normal. The contrast between these two babies and the breast-fed is certainly striking. The two babies receiving whole cow's milk, while not showing such distinctive blood and x ray findings, averaged between the two groups on clinical examination.

The chart (Fig 160) contrasts babies 8 and 9 and 11 and 12, two receiving lactose mixtures and two maltose-dextrin. The findings here are typical of the majority of the infants. To

Baby's No	1st Month	2nd	3rd	4th	5th	6th	7th	8th
8		Ca104 P 62 XR-0	Ca105 P 73 Ry? XR-0	Ca102 P 78 Ry? XR-0	Ca115 P 73 Ry? XR-0	Ca93 Cr? P 51 Ry? XR-0 HGT	Ca105 P 72 Ry? XR-0 HGT	
9		Ca130 Cr? P 72 Ry? XR 0	Ca105 P 78 Ry? XR?	Ca114 P 75 Ry? XR?	Ca108 P 59 Ry? XR? HG	Ca106 P 67 Ry? XR HG		
11		C 108 Cr? P 55 XR 0	Ca111 P 64 XR-0	Ca108 P 55 XR 0	C 96 Cr? P 62 Ry? XR 0 HGT	Ca100 Cr? P 49 Ry? XR-0 HGT	Ca106 P 55 Ry? XR-0 HGT	
12		Ca116 Cr? P 73 XR 0	C 103 P 56 XR-0	C 109 P 54 Ry? XR?	Ca102 Cr? P 49 Ry? XR? HGT			

L g m d  
 Cr Chromium  
 Ry R. Ray  
 HG Harrison's  
 Groove  
 XR X-Ray  
 Hgt  
 M "Whole Cow's  
 Milk

Fig 160—Same as Fig 159 on diets of milk with either maltose-dextrin or lactose addition

our great surprise most of the infants went through the six months' observation period with little abnormality on x ray examination and with normal blood chemistry and phosphorus. Physical examination apparently proved a more delicate diagnostic index than laboratory findings, for most of the infants at some time showed a mild rosary or Harrison's groove. But these findings were scarcely more marked than in some infants who had received routine treatment with cod liver oil. Again during the observations a mild rosary and Harrison's groove

might appear for a time and then improve spontaneously. Certainly not all the improvement seen in clinics and private practice during the first six months can be attributed to antirachitic prophylactics and remedies. Craniotabes proved perplexing. It occurred in some degree in all the infants during the early weeks. Then it disappeared. However, in the few babies developing true rachitic findings during and after the fifth month it reappeared. Apparently it is of no significance until this later period.

Babys No	1st Month	2nd	3rd	4th	5th	6th	7th	8th
15	Ca 10.9 P 6.0 XR 0		Ca 10.6 P 6.0 XR?	Cr? Ca 10 HG? P 5.1 Ry?	Ca 9.8 Cr+ P 4.6 Ry+? XR+ HG+?	Ca 9.0 Cr+? P 3.9 Ry XR+ HG	Ca 10.9 Ry? XR? HG?	Cr? Ca 10.5 Ry? HG? Ca 11.9 P 5.8 XR 0
	1/2 M + 1/4 BL		1/2 M + 1/4 BL		1/2 M + 1/4 BL		1/2 M + 1/4 BL	
19		Ca 12.5 P 4.2 XR 0	Ca 11.2 P 3.8 XR 0	Ca 11.6 P 6.6 Ry? XR 0 HG?	Ca 10.4 P 6.3 XR 0	Ca 10.0 P 6.1 Ry? XR ?		
	1/2 M + 1/4 BL		1/2 M + 1/4 BL					
20	Ca 11.8 Cr? P 10.9 XR 0	Ca 10.9 P 5.5 XR 0			Ry+? XR? HG+?	Ry+? XR 0 HG+?		
	1/2 M + 1/4 BL		1/2 M + 1/4 BL		1/2 M + 1/4 BL			

Legend  
Cr = Craniotabes  
Ry = Rosary  
HG = Harrison's  
Groove  
XR = X-Ray  
? = Mild  
+? = Fairly Definite  
M = Whole Cow's  
Milk  
BL = Beta Lactose

Fig. 161—Same as Fig. 160 on diets of milk with the addition of beta-lactose

The chart (Fig. 161) contrasts babies 19 and 20. They were both started on a milk mixture with beta-lactose addition, but later baby 20 received maltose-dextrin for some months. Here again the findings are similar to those in the preceding chart. Baby 15 is also shown in this chart, illustrating the effect of cod liver oil. This infant was started on a mixture of two thirds milk with low carbohydrate. He was the only infant on any milk dilution to develop pronounced clinical

symptoms Here the striking influence of cod liver oil is shown But what is even more surprising is the relatively small amount of the oil necessary From the middle of February 2 teaspoonfuls were given daily By March the laboratory and clinical signs showed decided improvement By April the baby had two teeth In this infant, simultaneously with the giving of the oil, a gram negative flora changed to gram positive and was so maintained after the oil was discontinued

Average Caloric Consumption Per Pound Body Weight																				
Baby's Age	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
2	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH
4	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH	WCH
6				54		40	48	57		61		48	WCH	54	64	57				59
8				52		48	42	55	60	70	63	60	56		59	58	62	69	49	58
10	63	70		51		50	48	58	60	63	63	64	58	60		58	68	65	66	49
12	55	50	55	51	62	55	46	60	59	54	76	55	59	50	68	66	67	65	60	61
14	60	53	56	50	60	60	47	57	50	85	52	64	51	63	69	63	62	49	60	
16	58	62	61	46	59	34	60	54	51	78	55	58	56	60	69	60	58	45	56	
18	53	66	57	44	53	51	50	56	50	45	82	55	54	55	53	59	45	56	46	52
20	48	59	55	48	51	52	48	54	46	44	77	54	52	52	60	55	56	58	46	50
22	50	55	53	42	58	54	49	46	54	77	52	55	50	58	55	53	54	45	48	
24	48	58	49	49	45	50	53		47		67	51	49	49	50	53	51	55	44	46
26	54		49	50	51	52	50		42		65	50		51	50	51	63		40	
28			56	47	51						62	53		50	54	50				
30				45																
32																				
34																				

Fig 162

In other attempts we were not able to obtain the same effect of the oil upon the flora

**Height and Weight**—All of the babies studied were definitely below the standards given as normal in the accepted tables Even those who were roentgenologically and chemically nonrachitic and who seemed in perfect nutritional condition weighed as much as 2 pounds below the accepted average Here, as in the infant just demonstrated one is uncer



tain whether the finding is due to "hospitalism" or to the lack of cod liver oil. If cod liver oil has the faculty of hanging several apparently unnecessary pounds on a normal infant, it may have an injurious as well as a beneficial effect.

*Caloric Intake*—The table (Fig. 162) shows the average caloric intake of each baby over periods of two weeks each. The table affords a comparison between the energy consumption of the majority of infants who showed very mild clinical rickets and those few showing no signs at all. It also shows the effect upon the caloric intake of increasing the concentration of the diet.

During the first few weeks the findings can be disregarded due to adjustments of the diet. Looking at the table as a whole, one sees at first no consistency in the findings. Or at any rate, there is no correlation in any one infant between the severity of the signs and the caloric intake. On the other hand, we have as controls baby 10 who was a perfect example of the breast-fed and baby 19 who was the most perfect clinically of the artificially fed. In these babies the caloric intake was not only decidedly less than in the others, but also showed a gradual reduction during each succeeding period. On the other hand, baby 11 who was practically normal clinically took a high amount. This baby was very small and seemed to require the amount given even for slight gain. He remained the smallest of the group at the time of his discharge from the hospital. Again baby 13, a breast-fed infant, took a surprisingly high diet. He was definitely below normal nutritionally and suffered for a time from generalized furunculosis. While it is difficult to formulate definite conclusions, one gets a certain clinical impression that the rachitic infants generally consumed more calories than were necessary for a normal gain in weight, or at any rate, they continued a high caloric consumption over a longer period of time with no spontaneous reduction in intake as did babies 10 and 19. For instance, baby 20 showed definitely more positive findings than did baby 19. The difference in caloric intake is apparent.

I always had the impression that babies on concentrated

diets automatically restricted their caloric intake to their requirements. This is definitely not so. Babies 1 and 2 on the most concentrated diet were frequently hungry and dissatisfied with the quantities offered. It will be seen in every case that when the diet was concentrated to higher caloric value, the infant invariably increased his caloric intake to a quantity greater than was necessary for a gain in weight. He did not adjust his food intake to his caloric need. In other words, concentrated diets lead to overfeeding. This may be of importance in the long continued usage of such formulae.

**Relations of the Elements of the Diet**—Rickets developed in decreasing severity in diets as follows

Whole cow's milk with 12 per cent carbohydrate

Whole cow's milk.

Whole cow's milk with 12 per cent carbohydrate appropriately diluted

Breast milk

The milk mixtures uniformly used showed a relation of carbohydrate to protein approximating that in human milk. With the exception of those in which overfeeding played a rôle, these formulae even in the absence of vitamin D were accompanied by rickets of only a mild type. These observations should be repeated with other formulae. Possibly the proportions of the mixtures may have been of some importance.

**Growth Curves**—The chart (Fig 163) shows the weight curves of four infants. One received cow's milk with 12 per cent carbohydrate, the second, a dilution of the above, the third, cow's milk with no addition, and the fourth, breast milk. Here again we see another manifestation of the different effects of these formulae. All these babies were subjected to monthly determinations of blood calcium and phosphorus as well as to repeated periods on the metabolism bed. In spite of these interruptions the breast fed infant pursued a relatively smooth and consistent course. The baby receiving the milk dilution was next. The baby on undiluted cow's milk fell below the standards of the others while the one receiving the high caloric mixture had a turbulent time. The

latter curve is also interesting in that it shows that a high caloric diet does not necessarily lead eventually to a large baby. While the growth of this infant was first rapid and far above normal, the curve eventually flattened out to approach the others at about the six-month period. Excessive feeding then could not obviate nature's plan for the eventual weight of this infant and he had to dispose of his extra food as best he could.

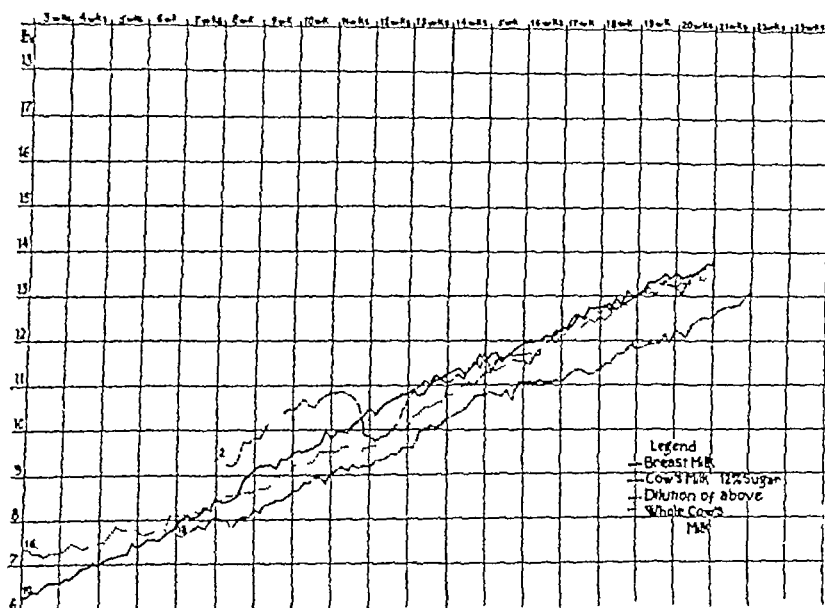


Fig. 163—The weight curves on diets of breast milk, cow's milk with 12 per cent carbohydrate, dilutions of the latter, and undiluted whole cow's milk.

If there is a dietetic factor in the prevention of rickets it probably is in the combination of proper calories and proper proportion of food elements existing in ideal form in human milk.

**Cod Liver Oil**—The fact that most of the infants went through six months in a hospital with no cod liver oil, no known source of vitamin D or ultraviolet light, and with rachitic findings of only a very mild type cannot be overlooked. Again the relatively small amount of cod liver oil

necessary to right the condition raises two questions Is the effectiveness of the oil influenced by physicochemical conditions in the intestine? In baby 15 (Fig 161) the laboratory and clinical response to relatively small amounts of the oil was striking Or is it true that cod liver oil and vitamin D are prescribed in unnecessary quantities and that we physicians and our patients are being exploited?

Medical men have undoubtedly known that many babies will not develop rickets during the first six months, even if given no antirachitic They have undoubtedly suspected that antirachitics may be given in unnecessary quantities and they have probably suspected that determinations of blood calcium and phosphorus as well as x ray examinations are not reliable indices as to the existence of rickets But for some reason or other the whole subject has been shrouded in more or less silence

**Summary** —To summarize this clinic, we have specifically raised the question as to why some babies develop signs of rickets in spite of a supposedly adequate dosage of vitamin D We have suggested that one factor might lie in the present-day methods of feeding which seem to us to lay undue emphasis upon the desirability of maximal size values

We have also raised the question and entered into a scientific study of the importance of the chemical effect upon the infant's intestine of some of the diets commonly used If calcium and phosphorus are absorbed more readily from an acid intestine, an increased incidence of rickets might be ascribed to present day methods of feeding because most of the popular formulae do not lead to high intestinal acidity The only carbohydrates which seem to do this are lactose and beta lactose and up to recently these have not been used extensively

In the 20 babies studied clinically with special reference to the development of rickets, there seemed little difference between those receiving maltose-dextrin and those receiving lactose Those on lactose and beta lactose may have shown slight clinical superiority but such conclusion is tentative until further chemical studies are completed

Much more apparent were the differences shown by babies receiving whole boiled cow's milk with 12 per cent carbohydrate addition, dilutions of this mixture, whole boiled cow's milk with no carbohydrate addition and breast milk. As none of these babies received antirachitics, it is presumable that a dietetic factor was also involved. Excluding the influence of the degree of acidity of the intestinal contents, this dietetic factor probably lies in the caloric content, concentration, nature and percentages of the food elements in the formula. Certainly in those few infants developing signs of rickets the severity of the condition in a general way paralleled the concentration of cow's milk and the excess of carbohydrate in the formula.

In this study a number of incidental observations were of interest.

It was somewhat out of the ordinary to note that most of the babies could spend six months in a hospital ward, receive no antirachitics and show rickets of only the very mildest type or often no rickets at all. In the babies with positive signs, cod liver oil was effective in smaller doses and over a shorter period of time than I had thought necessary.

In this study physical examination proved a far more delicate method of determining the early development of rickets than did the x-ray or determinations of blood calcium and phosphorus. Craniotables was of little diagnostic significance before the fourth or fifth month.

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